Symptoms of virus diseases in plants

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SYMPTOMS of virus diseases in plants

With indexes of names in English, Dutch, German, French, and Italian and 41 illustrations

The author is a virologist at the Institute of Phytopathological Research. This Institute is one of the autonomous foundations sponsored by the Dutch Ministry of Agriculture. The Institute was established in 1940 to study the efficient and economic control of pests and diseases of agricultural and horticultural crops.

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Preface

ADOLF MAYER, investigating more than 80 years ago an intriguing tobacco disease, chose a very original name to describe it: 'mosaic'. Nowadays we know of a considerable number of similar mosaics in the plant kingdom. But plant viruses are not solely known as incitants of mosaic diseases and, in fact, a great variety of other symptoms also due to virus infections have been described.

For years symptoms were the only means of characterizing virus diseases in plants. Thanks to modern biophysical, biochemical and serological research methods, however, more information has gradually become available concerning the intrinsic properties of the viruses themselves, thus enabling phytovirologists to distinguish these incitants of disease and the diseases they incite in a more unequivocal way.

Nevertheless, symptoms remain of considerable current interest. They are indispensable in the phytopathological evaluation of the effects of virus diseases and are usually the only means for recognizing quickly virus diseased plants in crops. Moreover, they still constitute the basis for the naming of plant virus diseases and their incitants. Thus terminology regarding symptoms of virus diseases continues to warrant critical consideration.

In the course of several decades a large collection of terms describing the phenomena of virus infections in plants has arisen. Authors publishing in English have contributed especially in this respect. Unfortunately, however, evaluation of these terms has not always been made. Since in other languages often no exact or convenient equivalents of the English terms were at hand, the originals have in very many instances been borrowed untranslated. These factors have led to a situation in which a systematic account of the data regarding symptomatology and its terms would be most welcome. Dr. Bos therefore deserves credit for undertaking the task of writing this book. That he sent the

manuscript to colleagues in various countries requesting for suggestions and critical remarks has also added greatly to its value. Thus the Index of names of symptoms with equivalents in five languages may promote an internationally uniform use of terms.

Of course no one can force the use of certain terms in a particular way. It is hoped, however, that plant virologists will take cognizance of its contents and that they will use it. Hence, a type of standardization will be achieved which will benefit communication in the field of plant virology.

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Wageningen, 1962

Author's Preface

The user of this publication should bear in mind that it is not meant to be a monographic literature survey. Only the best examples available to the author are given to illustrate the symptoms described.

I am aware that this book would only add to the confusion unless the names and definitions used are agreed upon by a good majority of plant virologists. For this reason copies of the manuscript were sent to several outstanding virologists all over the world, and I am greatly indebted for the many encouragements, suggestions and comments that were received. It is a real pleasure to acknowledge the help of so many virologists. It is impossible to mention personally all Dutch colleagues in the Institute for Phytopathological Research, in the Department of Virology of the Agricultural University, and in the Plant Protection Service at Wageningen, with whom I have discussed this publication. Special mention should be made of the useful suggestions and help of: Drs. C. W. Bennett, Salinas, Cal. U.S.A., R. Bovey, Nyon, Switzerland, M. Chessin, Missoula, Mont. U.S.A., R. W. Fulton, Madison, Wisc. U.S.A., P. Grancini, Bergamo, Italy, F. O. Holmes, New York, N. Y. U.S.A., D. D. Jensen, Berkeley, Cal. U.S.A., E. Köhler, Braunschweig, Germany, P. Limasset, Montpellier, France, F. Pelet, Nyon, Switzerland, G. S. Pound, Madison, Wisc. U.S.A., A. F. Ross, Ithaca, N.Y. U.S.A., H. H. Thornberry, Urbana, Ill. U.S.A. and H. A. Uschdraweit, Berlin-Dahlem, Germany. I discussed the subject personally with many of them. However, the final interpretation and definition of terms is my own responsibility. I will be glad to receive further suggestions and comments.

It is greatly appreciated that Dr. R. Bovey and Dr. F. Pelet, Nyon, Switzerland, contacted the French speaking plant virologists for the addition to the index of a complete list of French synonyms. Similarly thanks are due to Dr. P. Grancini, Bergamo, Italy and Dr. O. Lovisolo, Turin, Italy, for devising

a list of Italian synonyms. MR. J. VEERING, linguist of the Technical University, Delft, checked the linguistic qualities of the Dutch terms.

Several photographs have been generously provided by virologists, whose names are given under the illustrations concerned.

I am especially indebted to DR. D. D. JENSEN, Berkeley, California, DR. G. NYLAND, Davis, California and to DR. A. F. ROSS, Ithaca, New York, U.S.A., for their help with the English language.

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L. BOS

Wageningen, 1962

Introduction

Among plant pathogens, viruses occupy a unique place. In contrast with the parasitic organisms, they can enter the plant passively only through extremely small wounds. These may be produced, for example, by breaking of epidermal hairs by mutual contact of diseased and healthy plants, or by insects simultaneously carrying and introducing the virus. In this way the viruses reach the living plant cells. This introduction is called *inoculation* [1].

Only if the inoculated cells have *susceptibility* to the given virus, it becomes established in the cells' protoplasm and starts initiating biochemical changes. How this occurs is still unknown. The virus establishment and beginning virus activity is called *infection*. If the inoculated cell is not susceptible, inoculation does not result in infection. This total lack of susceptibility is known as *immunity*.

In the literature there appears to exist a tendency to use the term 'infection' for the sum total of the host-virus interactions resulting from the establishment of a virus in a susceptible cell. For this interaction the term 'disease' exists, however, with some emphasis on the host reaction.

The term infection points to the communication of an 'infectious disease' from one organism to another. Infection is prerequisite to the disease; it initiates the disease.

In susceptible plants abnormal processes incited by the virus lead to virus multiplication and to physiological changes producing functional and morphological abnormalities. These are perceptible at first in the inoculated cells, later on in surrounding tissues and often finally in the entire plant. Thus, the abnormalities are initiated at a cytological level, and develop into deviations visible to the naked eye. Since the plant or plant parts

1) The terms printed in italics are listed in the index with Dutch, German, French and Italian equivalents. Italics are only used where a defining discussion of the terms is given.

suffer from them, the changes are known as *disease symptoms* (Gr. symptoma = anything that has befallen one; from: sympiptein = to fall together) or pathological phenomena (Gr. pathos = suffering). The whole group of symptoms caused by a given virus or other pathogen are known as the *syndrome* (Gr. syn. = with, together; dramein = to run). They form the clinical picture of the *disease*. Biologically the disease starts at the very beginning of the host reaction. However, the disease becomes clinically evident at the moment this reaction causes changes perceptible to man. The period of time elapsing between inoculation and appearance of symptoms is called *incubation period*.

The severity of the pathological effect in a susceptible host depends upon its *sensitivity*. A low degree of sensitivity is known as *tolerance*. It may be associated with normal virus multiplication, but is characterized by mild symptoms. Extreme tolerance results in absence of a perceptible host reaction or *inapparency*. This absence of symptoms will be discussed more in detail in the next chapter.

A low degree of sensitivity or a low degree of susceptibility is often called *resistance*. This term especially refers to resistance to the virus, resulting in restricted or retarded virus multiplication, but it also indicates the associated mildness of the disease. There are several types and degrees of resistance. In practice, such as among plant breeders, the term is used to indicate the total of all host factors that cause the plant to escape infection or to withstand infection without adverse effects. Extreme resistance equals *immunity*, as does insusceptibility as mentioned above. Sometimes plants escape from disease and show resistance to natural infection because of *hypersensitivity* (cf. p. 51). Then sensitivity of cells infected upon inoculation is so high that a rapid death of these cells prevents further spread of the virus inside the plant.

The particular origin of virus symptoms generally makes them fundamentally different from those produced by parasitic organisms. Moreover, the latter disease incitants usually have a local action and often take part in the symptoms by means of a visible mycelium or characteristic sporulations and fructifications in, on, or in the neighbourhood of the infected parts of the host. Therefore, in contrast with virus diseases, they can be identified easily in many cases.

Thus, virus symptoms only are products of a plant metabolism upset by the virus. Obviously this host-plant reaction depends on the physiological condition of the host. Since this condition depends largely on host species and variety, age, nutrition, climate, the precence of another virus, etc., it is self-evident that all these factors determine the nature and severity of the symptoms produced. As a consequence, the virus symptoms are highly variable. Moreover, they often resemble more or less other physiological disturbances such as mineral deficiencies or abnormalities due to toxic agents, to overdosing of growth hormones used as weedkillers, or to some genetic disorders.

Before the symptoms are described in detail, some terms indicating special aspects of the host-virus relationship should be explained, such as those associated with the absence of symptoms and the sequence of symptoms. Also, the linguistic aspects of the naming of symptoms need to be set forth.

Absence of Symptoms

In the introduction mention was made of invisible biochemical changes which lead to visible abnormalities. Infection and virus multiplication, however, do not always lead to visible symptoms. This absence of visible symptoms is known as *inapparency*. JAMES JOHNSON (1925) was the first to direct attention to the presence of viruses in apparently healthy potato plants. Since then the problem of inapparent infection had considerable attention, especially in connection with the certification of virus-free propagating material. Inapparency, however, is also a quite general problem in the control of virus diseases, since hosts with inapparent infection may act as a focus of infection for neighbouring sensitive individuals, varieties or other crops. A review of literature, a great many examples and practical implications of inapparent infection are given by HILDEBRAND (1958).

Many viruses have hosts in which infection never causes visible symptoms. In plant virology this permanent type of inapparency is called *latency* (L. latere = to lie hidden). These susceptible hosts have no sensitivity, but do have extreme tolerance. They do not react visibly to the virus. Since the discovery of this phenomenon by NISHAMURA in 1918, such hosts have been called 'carriers'. The presence of virus, however, can be demonstrated by back-inoculation to sensitive hosts, by serology or by electron microscopy. Actually a number of new viruses have been discovered in experiments in which sap from symptomless plants induced symptoms in experimental hosts. Examples are dodder latent mosaic virus (BENNETT, 1944), and carnation latent virus (KASSANIS, 1954). The latter was discovered also to be latent in a number of potato varieties.

Many plant viruses have been associated with certain plant species, especially wild plants, for long periods. From an evolutionary point of view it seems probable that in these hosts these viruses have reached a state of equilibrium in which they are able to persist with the production of a minimum amount of injury (BENNETT, 1958).

In a recent discussion of a number of aspects of 'gradations between pathogenicity and commensalism in infections with plant viruses', BAWDEN (1958) proposed the term 'commensalism' for latency. This indicates the existing together in harmony of plant and virus.

In sensitive hosts there always is a certain symptomless period after infection. This *incubation period* may vary in length from some days to over a year, depending on virus, host species and conditions.

Sometimes virus symptoms may disappear temporarily; newly formed organs may be free of symptoms, but after some time symptoms may return. This phenomenon is commonly named *masking* and is often caused by environmental factors such as temperature. An instructive example is that of prune dwarf in Italian prune, which disease is masked at temperatures above 13° C (MOORE, personal communication) (fig. 1). If the disease is more or less masked permanently, it may be called *recovery* even though active virus is still present, such as in tobacco plants with tobacco ringspot (PRICE, 1932, 1936). The ultimate nature of this recovery phenomenon is unknown. It is clear, however, that total recovery can only be distinguished from latency by the presence of symptoms in older parts of the plant.

In literature on plant viruses the terms latency and masking, as two forms of inapparent infection, are commonly used in the above sense (cf. e.g. the recent German textbook on plant viruses by KLINKOWSKI and collaborators (1958)). In the whole field of plant pathology much confusion as to the exact definition of these terms exists. According to GÄUMANN (1945) 'inapparent' means a permanent absence of symptoms and 'latent' a temporary absence of symptoms. Thus in the case of cereal and grass smuts BUTLER & JONES (1949) speak of a latent infection. Here infection becomes apparent when the ears of individual caryopses are developing at the end of the vegetative life of the host. Since 'latent' literally means hidden or dormant (L. latere = to lie hidden) this seems not to be inaccurate. Presumably this is why even in plant virology the incubation period of a virus in the insect vector, i.e. the time elapsing from the uptake of the virus until the vector can transmit it to healthy plants, often is called a latent period. Therefore the agreements attained at a medical 'Symposium on latency and masking in viral and rickettsial infection' (1958) are not all applicable to plant virology. It was suggested that the term 'inapparent' be used for all infections without visible symptoms and 'latent' only for those inapparent infections which are chronic and are the result of a balance between host and virus. In this symposium it was proposed that the term 'masking' be dropped. Since this term is quite current in plant virology, however, and a distinction between permanent and tem-

Fig. 1. Masking of the symptoms of prune-dwarf virus in Italian prune induced by temperatures above 13° C. From left to right: (1) permanent low temperature, (2) temperature at first below and later above 13° C, (3) temperature at first above and later below 13° C, (4) constant high temperature. (Photo courtesy Dr. J. D. Moore, Madison, Wisconsin).

porary absence of symptoms is needed, it will be difficult to eliminate this name. As it literally means unrecognizable but still visible, the term 'masked' seems to be rather incorrect, however. To conclude, the distinction between inapparency, latency and masking is more a matter of usage and agreement than of literal meaning.

The concepts of inapparent, latent and masked infection have a very relative meaning. Theoretically a really latent infection may be possible, e.g. if virus multiplication could take place by utilising cell materials which are present in excess of those needed for normal plant metabolism. As yet no exact data on

this possibility are available, however. Generally virus multiplication cannot take place without any influence on plant physiology. This influence may be so very small that it is not noticed.

Now the problem arises of distinguishing between apparent infection inducing visible symptoms and inapparent infection being entirely imperceptible. For everyone acquainted with living nature, however, it will be clear, that it is impossible to draw a distinct borderline between visible and invisible reactions of the plant to virus infection. In the same way it is impossible to delimit abnormal and normal, pathological and healthy, in plant growth (cf. KÜSTER, 1925; BLOCH, 1954).

The difficulty in distinguishing between diseased and healthy plants is especially demonstrated by results of potato virus research. After JAMES JOHNSON (1925) showed that sap from American potato plants, whether seemingly healthy or diseased, always produced virus diseases when inoculated to tobacco and some other Solanaceous species, this problem got much attention. A good example is given by the potato virus S, which was discovered serologically by DE BRUYN OUBOTER (1952) and studied extensively by ROZENDAAL & BRUST (1955). By means of serological tests it was shown that this virus has a very high incidence and that several potato varieties are almost 100% infected. A few potato varieties appeared to produce a slight mosaic, whereas most varieties developed nothing but very faint symptoms, that only could be perceived when an accurate comparison was made of infected and virus-free plants. The variety 'Industrie', being generally infected, nevertheless has a flourishing appearance. Usually the first visible reaction is a depression in yield. In the same way KREITLOW *et al.* (1957) demonstrated that Ladino clover, after infection with a mixture of bean yellow mosaic virus and lucerne mosaic virus, often shows a decrease in yield, even in cases where symptoms are scarcely noticeable. These examples clearly demonstrate that the distinction between inapparent and apparent infection is only arbitrary and that the position of the borderline between them often depends on the accuracy and the tools with which the reaction of the plant is studied.

Sequence of Symptoms

One way of distinguishing virus symptoms is on the basis of the course of infection and the associated sequence of symptoms.

After the incubation period the first host reaction mostly occurs at and around the place of inoculation. These *local symptoms* may be more or less shock-like and restricted to the inoculated cells and their immediate neighbourhood. Those defined areas of visibly diseased tissue, or *lesions*, occurring locally at the site of virus entry are generally called *local lesions* (fig. 2, left). The morbid effects may consist of discolouration, desiccation or even death of tissue and will be discussed in detail in the sections concerned.

It should be well understood that as a matter of agreement and usage the adjective 'local' is added to point to the fact that the lesion has been developed at the 'locus' or site of inoculation. It does not refer to the localization of the reaction to a restricted area of tissue, since this is already included in the meaning of the word lesion. Lesions may also occur in plant parts systemically invaded by the virus (see below).

If the local reaction to virus inoculation is necrotic, further invasion of the plant by the virus is often prevented (*hypersensitivity*, cf. p. 51). Usually, however, the virus becomes systemic; it spreads internally through the inoculated leaf and finally through the whole plant. This may occur rather rapidly, especially after the virus reaches the vascular bundles. After some time the young, still developing plant parts not inoculated directly develop *systemic symptoms*. In some cases local symptoms become visible after the production of systemic symptoms. Very often only systemic symptoms occur and no abnormalities are produced in the inoculated leaves.

Sometimes systemic infection also leads to the development of localized areas of visibly diseased tissue. This development of *systemic lesions* (fig. 2, right) might be due to a low virus concentration in the transported phloem contents, giving rise to a localized reaction at those spots where infectious units succeed in establishing new multiplication centers.

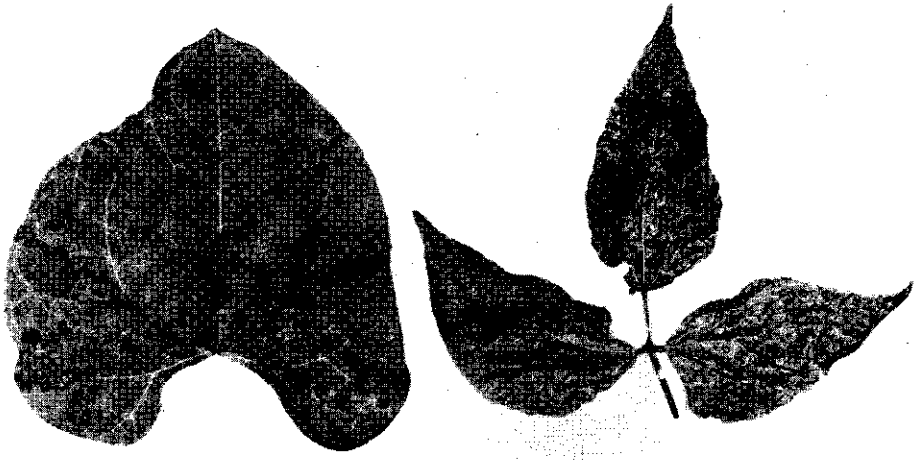


Fig. 2. Local and systemic lesions in bean leaves (Phaseolus vulgaris) after infection with an isolate of the early-browning virus of pea. Left, local lesions consisting of necrotic rings and ringspots in the inoculated primary leaf; right, necrotic speckling and ringspotting in a systemically infected trifoliate leaf.

Another distinction is that of *primary* and *secondary symptoms*, indicating their chronological sequence. Mostly local lesions are the primary symptoms, whereas the systemic symptoms are secondary. If local lesions or other local symptoms are absent, the first systemic reaction, such as e.g. vein clearing, may be considered as primary symptoms. Therefore the terms 'systemic symptoms' and 'secondary symptoms' are not equivalent, as is the case with the names 'local symptoms' and 'primary symptoms'.

It should be borne in mind, that the adjectives primary and secondary have a very general meaning as such, and are sometimes applied in another sense than the above. In seed-borne diseases (e.g. virus diseases of potato and common bean mosaic) they are used to indicate the primary and the secondary stage, respectively, of a continuous systemic infection. The primary stage (e.g. primary leaf-roll of potato) develops after an infection during the season and may already show systemic symptoms. The secondary stage (e.g. secondary leaf-roll of potato) develops from the infected seed or vegetative propagating material. For a discussion cf. OORT (1959).

In view of their different origin, it is evident that local and systemic symptoms generally differ fundamentally. In both cases, the tissues concerned at the moment of infection differ physiologically, such as in age and differentiation. The inoculated leaf is older than the one invaded systemically while still developing, whereas the leaves in the neighbourhood of the growing point are still in a meristematic stage.

Even the apical and basal parts of the same leaf may differ in their reaction to virus infection, since a leaf matures earlier at the apex than at the base. Therefore, local lesions may originate in the top part of the leaf and mosaic and other symptoms in the lower part, if these leaves were partially differentiated before virus entry.

The difference in physiology of inoculated and systemically infected parts of the plant also may lead to a distinction between an acute and a chronic phase. The *acute phase* may occur soon after inoculation, is more or less shock-like, and is therefore often called *shock phase*. This phase is characterized by severe symptoms that sometimes lead to death of the host. More commonly the host survives and a *chronic phase* follows. This phase often is characterized by some *recovery*, i.e. newly developing parts develop less severe symptoms than those evident during the acute phase. Even total recovery may result.

Acute and chronic phases even may alternate in the same plant. A typical example is the 'Echte Ackerbohnenmosaik' virus in broad bean. Infected plants show a periodical course in severity of symptoms. Groups of leaves with severe symptoms alternate with leaves with mild or no symptoms. Here, however, the periodical shift in severity of symptoms is not especially due to a variation in development of the systemically infected parts, but to a variation in virus concentration as was shown by PAUL & QUANTZ (1959). The reason for this variation in virus concentration is not yet known. The periodical shift in severity of symptoms is of great importance, since it may be comparable to the concentric *ringspotting* (p. 42) where zones of severely and moderately diseased tissue alternate. How far a decrease in virus concentration some time after infection plays a role in the development of a chronic phase, is not exactly known. There are examples of recovery, where a considerable decrease in virus concentration has been reported. This decrease amounted to

5–10 times in tissue of tobacco recovered from tobacco ringspot in comparison to apparently diseased tissue (PRICE, 1936).

In view of the effect of environment on symptom expression, as has been pointed out in the introduction, it is obvious that changes in external conditions such as in light intensity and in temperature also may induce shifts in severity of symptoms, may cause a sequence of different symptoms, or even may lead to masking.

There may also be a progression in severity of symptoms, such as a progressive debilitation or a progressive deterioration. This progressive effect is known as *decline*, and may be gradual (slow decline) or rapid (quick decline). The phenomenon is quite common in fruit tree virus diseases, such as Western-X in peach, cherry etc. (REEVES *et al.* 1951) and citrus tristeza (or 'citrus quick decline') (BENNETT & COSTA, 1949). Especially in the latter disease it has been demonstrated that this decline is due to a progressive phloem necrosis immediately below the bud union in sour orange rootstocks grafted with infected sweet orange scions (for a survey cf. SCHNEIDER, 1959).

It should be stressed here that virus symptoms are not restricted to young and growing plant parts, as might be inferred from the above text. ESAU (1948) has pointed out that, although young plant organs are relatively highly susceptible to virus infection, fully formed plant parts also may develop symptoms upon inoculation. She gave some examples.

In older leaves systemic symptoms also may develop if sufficient virus movement into such leaves is assured.

The seemingly relatively low sensitivity of mature plant parts to virus infection is easily understood, since virus multiplication, being the essential preceding stage in initiating symptoms, entirely depends on physiological activity, presumably, especially the nitrogen metabolism. Moreover transport of assimilates, and therefore also of viruses, to full grown leaves is restricted, hence such leaves may escape systemic infection. Also since old leaves are often resistant to the establishment of infection, such leaves may not become infected unless a considerable amount of virus moves into them. Thus, for the same reasons that lead to mature plant resistance (BERCKS, 1951; BEEMSTER, 1958), there exists a resistance of mature plant parts to virus infection. However, this mature plant resistance differs considerably according to the virus.

Naming of Symptoms

The word symptoms is used to indicate the visible effects on the plant of infection by a pathogen. These effects may be named in terms indicating the final products, e.g. the 'yellow edge' and the 'mottle' of the leaf. The effects may also be named in terms indicating the processes altering the appearance of the diseased plants, e.g. 'edge yellowing' and 'mottling'. Both approaches lead to a different naming of symptoms: yellow edge – edge yellowing, mottle – mottling.

Since static situations seldom if ever occur in living material, the present author is inclined to prefer the use of the processes as a basis for naming symptoms. Moreover, the terms 'yellow edge', 'mottle', 'stunt', 'wilt', 'yellows' etc. are commonly used to indicate the diseases rather than the symptoms. On the other hand the names of symptoms are code-words more or less, and thus should be short. Therefore many authors prefer names as 'mottle' and 'wilt'. Moreover, no acceptable short term exists for the processes underlying some abnormalities, such as 'mosaic'. For these reasons, both ways of naming symptoms are used in the literature on plant viruses, often in combination. Finally there are some terms indicating both the process and the final product, such as 'malformation'. Possibilities as to the best way of naming symptoms differ also according to language, as will appear in the index.

Description of Symptoms

Any organization of data should be based on a system. The same holds for a classification of virus symptoms, for it would enable the division of symptoms into groups, a delimitation of specific symptoms, and an eventual definition of names and terms. The classification used in this paper will have a more or less ontogenetic basis, the most important criterion being the way in which the symptoms develop.

All virus symptoms originate in biochemical deviations. For a survey of literature on the biochemistry of plant virus infection cf. PORTER (1959). Actually, only very little is known of the biochemical background of the pathological processes. Some of the biochemical changes are visible themselves, such as the decrease in chlorophyll content and the increase in the concentration of carotenes and xanthophylls leading to colour deviations. From exact biochemical investigations on tobacco mosaic diseased and healthy White Burley tobacco plants showing these differences, we also know that the virus induces an evident increase in malic acid and a striking decrease in succinic acid content (VENEKAMP, 1957). How far these and other changes in organic acids, being only detectable by analytical procedures, will be of practical use in diagnosis, is difficult to predict. Moreover, since biochemical composition of normal plants, e.g. of organic acids, is very variable depending highly on growth conditions (e.g. VENEKAMP, 1959), it will be difficult to use quantitative composition of a single plant as an indication of virus infection. This has been demonstrated for sugar beets infected with yellows virus (JERMOLJEV & PRŮŠA, 1958) and for potatoes infected with virus X, virus Y, leaf-roll virus or stolbur virus (JERMOLJEV, 1959, personal communication). A thorough discussion of these patho-physiological phenomena would go beyond the scope of this publication.

After being instigated by cell metabolism, usually the symptoms are initiated in the anatomy of the plant. Commonly, the

visible pathological phenomena start at a cytological level, making the anatomical study of virus-diseased plants of great importance. Therefore the cytological disorders will be discussed in section I of this chapter.

The macroscopic deviations can be devided into two groups. The first group of aberrations directly result from anatomical abnormalities. They will be discussed in section II on growth reduction, section III on colour deviations, section IV on water deficiency, and section V on necrosis. The succession of these sections is more or less arbitrary. The abnormalities of the second group are due to a disorganization of usually normal cells and tissues. Cork formation, to be discussed in section VI, in itself is not abnormal, except in its manner and site of initiation; it belongs more to organizational disturbances than to purely anatomical abnormalities. The malformations listed in section VII are especially caused by disorganization.

In section VIII a discussion is given whether virus particles themselves, either directly or in the formation of inclusion bodies, can be considered symptoms. Section IX represents phenomena due to secondary causes introduced by a preceding virus infection.

Evidently nature cannot be captured in a generally acceptable system. Therefore it is not possible to classify virus symptoms perfectly or to separate the groups of symptoms completely. In the same way on p. 19 it appeared to be impossible to delimit the normal and the abnormal, the healthy and the pathological.

I. Cytological deviations

Symptoms of virus diseases in plants are initiated in the cells. The earliest visible changes are therefore of cytological nature perceptible with the microscope only in the anatomy of the plant. The cell deviations may be merely of structural, but also of chemical nature. Evidently there is an association of chemical and structural changes. The chemical changes are sometimes visible directly, especially those concerned with pigments, or are visible after staining with certain dyes or certain colour reagents. Since the microtechnics tracing these chemical changes are often called histochemical, the deviations concerned are sometimes called 'histochemical deviations'. It should be kept in

mind that these deviations, although especially obvious in tissues, are of purely cytological origin. A survey of literature on anatomical aspects of plant virus disease problems was given by ESAU (1938, 1948, and 1956).

Not much is known of the influence of viruses on the shape of cells. Important is the effect of viruses on size and number of cells. The term *hypertrophy* (Gr. hyper = over, above; trephein = to nourish) is used for an abnormal increase in size of cells, for abnormal cell enlargement, but is also used for abnormal enlargement of organs (cf. p. 79). The abnormal increase in number of cells is called *hyperplasia* (Gr. plassein = to mold). If the increase in number of cells is almost unlimited, such as in tumorous growths, the term (cell)*proliferation* is used now and then (cf. p. 59). The term *hypoplasia* (Gr. hypo = under, less than ordinary; plassein = to mold) refers to a development of fewer and/or smaller cells, but is also used for underdevelopment of organs. The term *atrophy* (Gr. a = not; trephein = to nourish) is used for a completely arrested development of cells or organs, for a total lack of cell multiplication and cell enlargement where it should occur in the normal development of the plant.

The effects, just mentioned, on size and number of cells often lead to malformation of organs. Certain viruses even influence differentiation of cells in growing points. These correlation or development disturbances will especially be discussed in the section on malformations.

A peculair structural deviation is the *excessive formation of tyloses* in the xylem vessels of grape vine affected with 'Pierce's disease' (ESAU, 1948b). This leads to growth reduction and wilting of the vine. The tyloses are outgrowths from the living cells of the wood parenchyma and medullary rays. These outgrowths pass through the pits in the vessel wall and swell up into thin-walled bladders in the lumen of the vessel.

Cork formation is of structural as well as of histochemical nature. Since the abnormal cork formation leads to a number of macroscopical abnormalities it will be discussed in a separate section.

Loss of turgidity and total collapse of cells are quite common in virus diseases. They are caused by *water deficiency* and *necrosis* (Gr. nekroun = to make dead, mortify) (dying) of cells. For details see the sections concerned.

Some chemical changes are visible directly, such as the effects on pigments in the cell. Destructive or depressive effects upon the chloroplasts lead to, or are caused by a decrease in chlorophyll content, perceptible as a lessening of the green colour. Because of lack of chlorophyll the presence of xanthophylls and carotenes becomes evident, leading to a yellowish colour. An abnormal increase in xanthophylls and carotenes may contribute to the yellowing. Also the concentration of anthocyanins in the cell may be affected by viruses. As a consequence abnormal red or even purple colours may occur in the leaves or colour deviations may occur in the flowers. In the case of death of tissue usually dark-coloured melanins are produced. These colour effects will be discussed in the section on colour deviations.

Some other typical (histo)chemical changes should be mentioned here. They are to a certain extent of diagnostic value. The first deviation, *gummosis*, is the production of reddish-brown gum-like substances. This is for example very characteristic for white clover mosaic in petioles and stems of French beans. The deposits of gum occur especially in groups of cells of the interfascicular parenchyma (Bos, unpublished). The gummosis, however, is often part of, or accompanied by, necrosis. Externally these effects may be visible as diffuse greyish internal discolourations.

A second well-known chemical deviation is the abnormal *accumulation of starch* in the leaves. This phenomenon is characteristic of a number of viruses that are generally accepted to occur especially in the phloem of the vascular bundles, such as *in pea leaf roll, potato leaf roll* and sugar beet *yellows*. This accumulation of starch can be easily demonstrated by means of iodine potassiumiodide after removal of the chlorophyll by means of alcohol.

For a long time the reduced transport of starch, especially in potato, was assumed to be due to necrosis of the phloem (cf. QUANJER, 1913). From the work of HENKE (1957) on sugar beet yellows, it appears that the transport of carbohydrates is disturbed by an abnormal activity of phosphatase. According to KLINKENBERG (1945) the anatomical disorders can be observed only after the transport of carbohydrates has been hindered.

At present the *excessive formation of callose* in the phloem of

stems and tubers of leaf-roll-diseased potatoes, before necrosis occurs, is of much practical and scientific interest. Whereas the sieve plates in phloem vessels of healthy plants are only covered with a thin layer or small plug of callose, the vessels of diseased plants are filled with a large quantity of callose. Even entire cells may be filled (VON BREHMER & ROCHLIN, 1931). This callose can be easily stained with resorcin blue or any of a number of other stains, even in potato tubers at a certain physiological stage after harvesting (Igel-Lange test, cf. SCHUSTER, 1956). The possibility of using the abnormal callose production for diagnostic purposes was reported somewhat simultaneously by IGEL & LANGE (unpublished but patented), BAERECKE (1955), HOFFERBERT & ZU PUTLITZ (1955), MOERICKE (1955), and SPRAU (1955).

A last and peculiar chemical deviation is the *defective lignification* of xylem and tracheids of apple, especially the variety Lord Lambourne, as a result of infection with the 'rubbery-wood' virus. Cross sections of diseased branches, after staining with phloroglucinol and hydrochloric acid, show large light-coloured islands in which the cell walls are thickened with cellulose instead of being lignified (BEAKBANE & THOMPSON, 1945). This leads to an extreme flexibility of the branches, which are 'rubbery' and 'cheesy'. This abnormality has been described as the *rubbery wood symptom* (PRENTICE, 1950b). Older trees develop a 'weeping' habit as the small branches bend under their own weight and under the weight of the crop.

The presence of virus particles and *inclusion bodies* in the host cells will be discussed in a separate section (VIII).

II. Growth reduction

A great many viruses induce a nonspecific, general reduction in growth vigour (cf. also group IX). Such plants remain smaller than normal in all their dimensions (fig. 3). When this is striking we speak of *dwarfing* or *stunting*. Morphologically, these plants can be normal, all the organs being underdeveloped proportionally. However, the phenomenon is often accompanied by other symptoms. The name has been applied improperly in some cases such as 'Rubus stunt' for a witches' broom disease of raspberry (PRENTICE, 1950a) (cf. also p. 76).

Fig. 3. Growth reduction in bean (Phaseolus vulgaris) as a result of infection with bean yellow mosaic virus; right, healthy plant.

When a plant becomes infected at a late stage of development, only the extremities of the branches or the top of the plant are stunted, e.g. in the case of 'pea stunt' (HAGEDORN *et al.*, 1959). (For the formation of rosettes, being a different phenomenon, cf. p. 72).

Often the growth reduction leads to production of small fruits. This sometimes may be very striking, such as in 'little cherry' of sweet cherries, where the known symptoms are confined to the fruits. At picking time infected fruits are only half as large, or less, than are normal ones (FOSTER *et al.*, 1951). In connection with witches' broom growth a reduction in size of leaves rather

commonly occurs, e.g. in 'little leaf' of brinjal (THOMAS & KRISHNASWAMI, 1939). Since here the symptom is due more to a disturbance in growth proportions than to a general growth reduction, this phenomenon preferably should be grouped among malformations (p. 76).

Growth reduction frequently occurs without accompanying symptoms. Often the reduction in gross plant size is not conspicuous, but the disease effect is finally noticed in a *yield reduction*. This may be due to the production of abnormally small fruits, as mentioned above, or to the production of less total weight of the plants than normal. Generally this is the most important economic aspect or virus diseases. Since size and yield of plants also depend on a number of other factors, such as nutrition, effects on size and yield are generally difficult to recognize as virus symptoms. Moreover this is a striking example of an area where there is no borderline between normal and abnormal.

III. Colour deviations

Changes in colour are very common in virus-diseased plants. These disorders are basically the same for leaves, stems and fruits. Since leaves form by far the majority of the plant surface, their colour deviations attract attention and have been studied extensively.

The colour changes are mostly due to chlorophyll disorders, such as a delayed or a decreased chlorophyll production. This is the reason why mosaic symptoms may tend to disappear after some time. Degeneration of chloroplasts may also occur, as has been shown by ESAU (1944) in full-grown leaves of beet infected with beet mosaic virus. The decrease in chlorophyll content results in a pale-green or yellowish colour. This phenomenon is known as *chlorosis* (Gr. chloros = light green). Usually other anatomical disorders, such as spherical shape of the palisade cells and delayed formation of intercellular spaces in the mesophyll, are also involved. Both lead to a decreased thickness of the discoloured parts of the leaf, often contributing to a further effect on the green colour. According to the survey of ESAU (1938, 1948) the picture differs with virus and host. In extreme cases

of chlorosis all pigments may be absent, a condition sometimes called *blanching* or *bleaching*.

Because of a lack of chlorophyll the presence of carotenes and xanthophylls becomes evident in chlorotic tissue, causing this tissue to show a *yellowing*, especially in those plants having high natural content of these pigments. Sometimes there may be also an increase in content of carotenes and xanthophylls contributing to the yellowing, such as in leaves of tobacco plants infected with tobacco mosaic virus (VENEKAMP, 1957). It should be kept in mind that the terms chlorosis and yellowing, although generally referring to associated phenomena and therefore often being used as equivalents, indicate basically different processes.

Anthocyanins are involved in abnormal *reddening* or sometimes *purpling* of plant parts and also in colour deviations in the flowers. The underlying biochemical changes are not yet known. Since anthocyanins are related to sugars, disturbances in sugar metabolism may be involved. This is further suggested by the fact that reddening and purple discolouration are rather often associated with diseases such as potato leaf roll and barley yellow dwarf in oats. These diseases, caused by viruses occurring especially in the phloem, are generally characterized by a disturbed carbohydrate metabolism.

In necrotic tissue the production of dark-coloured melanin-like substances results in *browning* (fig. 15) and *blackening*. If the necrosis is very superficial, such as in the epidermis, the colour effect may be that of *bronzing*. If the death of tissue is caused by rapid desiccation, the production of melanins may be prevented. Then the dead dry tissue may have a silvery grey or whitish colour. Colour changes due to necrosis and to desiccation will not be discussed in detail in this section.

a. Colour deviations in leaves

The terms *chlorosis* and *yellowing* generally refer to an even discolouration, regularly distributed over the entire plant, the entire leaf, or sometimes part of the leaf or the venation. If it is reported that a plant shows chlorosis or yellowing, this means that the entire plant or especially the younger parts show a general chlorosis or a general yellowing (cf. also some names as 'iron chlorosis' and 'lime-induced chlorosis' for deficiency

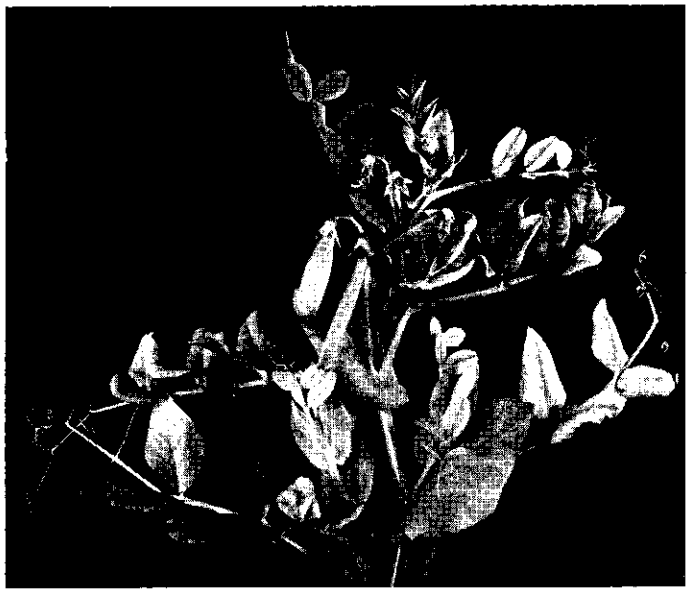


Fig. 4. Chlorosis and yellowing in pea caused by 'tip yellows' virus of peas (pea leaf-roll virus). (After HUBBELING, *1954).*

diseases). This type of leaf disorder is a typical symptom of some well-known diseases as 'sugar beet yellows' and 'aster yellows'. Since the symptom especially appears in leaves developing after infection, it may be restricted to the tips of the plants or of the branches, e.g. in 'tip yellows of peas' (pea leaf roll) (fig. 4).

Chlorosis and yellowing may also be restricted to certain parts of the leaves, giving more or less the impression of an 'arrested' or 'unfinished' distribution of the discolouration over the lamina. Examples are the chlorosis of leaf tips of barley plants infected with barley yellow dwarf virus and the *edge chlorosis* or *edge yellowing* in strawberry leaves, caused by the 'strawberry yellow edge' virus (fig. 5). Both the discolouration of the leaf tips and of the leaf edges tend towards a more general discolouration. Another type of 'unfinished' discolouration is that restricted to the venation, known as *vein chlorosis* and *vein yellowing*. The discolouration may spread to a certain extent into the tissue

Fig. 5. Edge chlorosis and edge yellowing in Royal Sovereign strawberry, affected by the 'yellow edge' disease. (Photo courtesy Mr. T. N. Hoblyn, editor J. Pomology and Hort. Sci.).

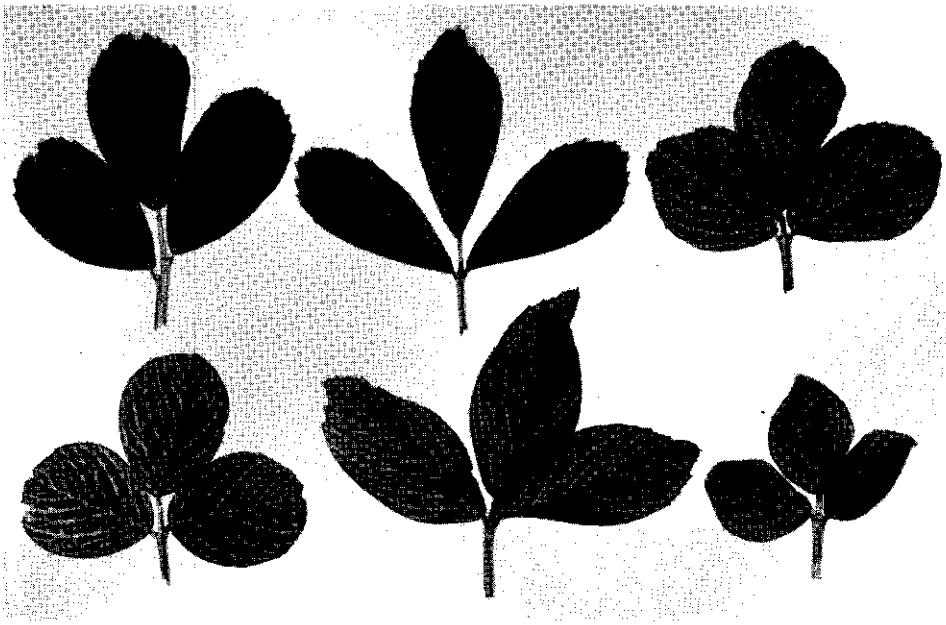
adjacent to the veins, but the pattern is always very regular. Good examples are the 'vein yellowing' of lucerne, var. Du Puits, caused by the tip yellows virus of peas (VAN DER WANT & BOS, 1959) (fig. 6), and the 'yellow net' disease of sugar beet (SYLVESTER, 1948).

There seems to be also a close ontogenetic relationship between the general chlorosis and general yellowing and the 'unfinished' types. 'Aster yellows', 'sugar beet yellows', 'vein yellowing of lucerne' and 'sugar beet yellow net' are all caused by persistent insect-borne viruses. The persistent viruses are known to occur especially in the

phloem and to induce disturbances in the vascular system, particularly in the phloem (ESAU, 1956). Chlorosis and the related yellowing are typical phenomena in diseases caused by persistent viruses and may be associated with disturbances in the vascular system. That there exists a gradation between general chlorosis and general yellowing and vein chlorosis and vein yellowing is further suggested by the fact that the tip-yellows virus of peas induces chlorosis and yellowing in peas and only vein yellowing in lucerne.

Related to the phenomenon of vein chlorosis and vein yellowing is the symptom of *vein clearing*, which differs in that the veins become translucent (ESAU, 1933, 1948) rather than chlorotic or yellow. The translucence is presumably due to a delayed formation of intercellular spaces. This close packing of parenchyma cells may be due to cell enlargement and cell division, such as in

Fig. 6. Bright vein yellowing in lucerne leaves, var. Du Puits, caused by tip-yellows virus of peas. (After VAN DER WANT & BOS, *1959).*



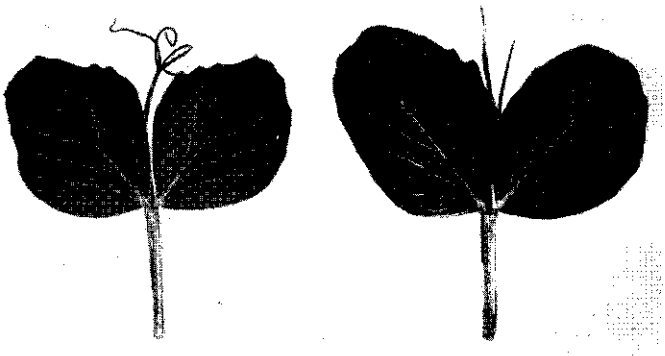


Fig. 7. Vein chlorosis in pea leaves, the primary symptom of infection with pea mosaic virus. Note that the veins are not translucent but only chlorotic. Left, healthy leaf.

curly-top beet leaves (ESAU, 1933). According to this author in this disease it may be accompanied by chloroplast degeneration. This makes it often hard to distinguish between vein chlorosis and vein clearing. In many diseases vein chlorosis and vein clearing are a first or early symptom and are often temporary (fig. 7).

In contrast to the more regular colour changes just mentioned, the discolourations may also be irregularly distributed over the whole lamina. This phenomenon is called *variegation* and is very common in plant virus diseases. Several types of variegation can be distinguished. Such a differentiation may be helpful in reducing the confusion existing in the description of these colour deviations and in the nomenclature of the virus diseases concerned. This differentiation can be based on differences in shape and sometimes in size of the discoloured areas, in sharpness of the boundary between dark- and light-coloured parts of the leaf and in distribution of the discolouration over the lamina. As far as the colours are concerned, the variegation may be chlorotic or even yellow and white. Sometimes even degrees in brightness can be indicated. Usually the types of variegation are described on the basis of combinations of these differences. The most

Fig. 8. Mosaic in Abutilon striatum caused by the 'abutilon mosaic' virus.

important types are the mosaic symptoms, flecking and associated phenomena, mottling, and the line-pattern symptom.

The term *mosaic* covers a group of symptoms that are all characterized by a mixture of irregularly shaped dark and light green or yellow areas on the lamina. Moreover, those parts of the leaf having different colour are sharply bordered, as is the case in surface decorations, made by inlaying of small pieces of glass, stone or other material, from which the term mosaic has been derived. Since MAYER (1886) introduced the name mosaic for the tobacco disease, of which he for the first time scientifically proved its infectious nature, mosaic symptoms are generally known. For a long time the names mosaic disease and plant virus disease were synonyms. It should be borne in mind that gradually oftentimes the term mosaic got applied in the broad sense of variegation, unproperly, however.

Very typical examples of a mosaic, with a uniform distribution of the light- and dark-coloured areas over the entire leaf, are the well-known 'abutilon mosaic' (often called 'abutilon infectious variegation') (fig. 8), and 'tobacco mosaic'. Here the irregularly shaped, light- and dark-coloured parts of the leaf are sharply defined and the borderlines, often being the small veins, are straight-lined.

The nature of the discolouration may be indicated in using the adjectives chlorotic or yellow.

There are some erroneous names in connection with the description of the discolouration connected with mosaics. The name 'abutilon infectious chlorosis', which is often used, is confusing since it suggests a general pallor of the green colour. The name 'abutilon mosaic' is preferable. In this plant species the mosaic may be chlorotic or yellow.

In the literature a difference of opinion exists as to the desirability of terms like 'aucuba mosaic' and 'calico' for yellow or very bright mosaics. The term *aucuba mosaic* refers to the variegated leaves of *Aucuba japonica* var. *variegatum* and is sometimes used for naming virus diseases, such as 'potato aucuba mosaic'. The term is inadequate, however, since the variegation in aucuba not always is a mosaic but may also consist of flecks (cf. p. 41). Moreover, the name may suggest a disease being caused by a virus, inducing mosaic in aucuba (cf. USCHDRAWEIT, 1958), as is the implication of a name like 'peach mosaic'. This argument especially holds for the application to a yellow mosaic in woody plants. Since the terms yellow mosaic or yellow flecking sufficiently cover the phenomenon, the term 'aucuba mosaic, had better be omitted. The same is true for the term *calico*, which is sometimes used for mosaic in which the brilliant yellow or sometimes

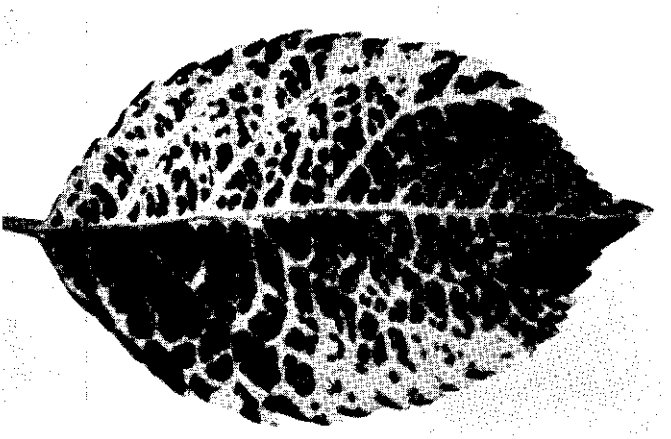
almost white parts of the leaf dominate. Examples are 'potato calico' caused by the lucerne mosaic virus (BLACK & PRICE, 1940), and 'peach calico' (BLODGETT, 1944). The name 'calico' is confusing since in America it actually is the name of brightly printed cotton cloth, having various colours, whereas in England it refers to plain white cotton cloth. The only literal meaning of the word is that the material had been imported first from Calicut, India. Therefore the term 'calico' to indicate a symptom should be omitted.

The size of the areas of different colours may be expressed in adjectives like coarse and fine and other words. Sometimes the discoloured areas have a certain shape. For example in monocotyledonous plants, which have parellel-veined leaves, the light coloured parts tend to become elongated. This results in a streak mosaic or stripe mosaic. The symptom may as well be called *streaking* or *striping* (cf. also p. 42).

The term 'streak', such as in 'cocksfoot streak' or 'pea streak', is confusive and insufficient. The name does not indicate whether the streak is chlorotic or yellow, such as in 'cocksfoot streak', or is necrotic, such as in 'pea streak' (cf. p. 54).

Other types of mosaic can be distinguished in which the mosaic patterns are confined to definite areas of the leaf. In *vein mosaic*

Fig. 9. Yellow vein mosaic of apple caused by 'apple mosaic' virus. (Photo courtesy Plant Protection Service, Wageningen).



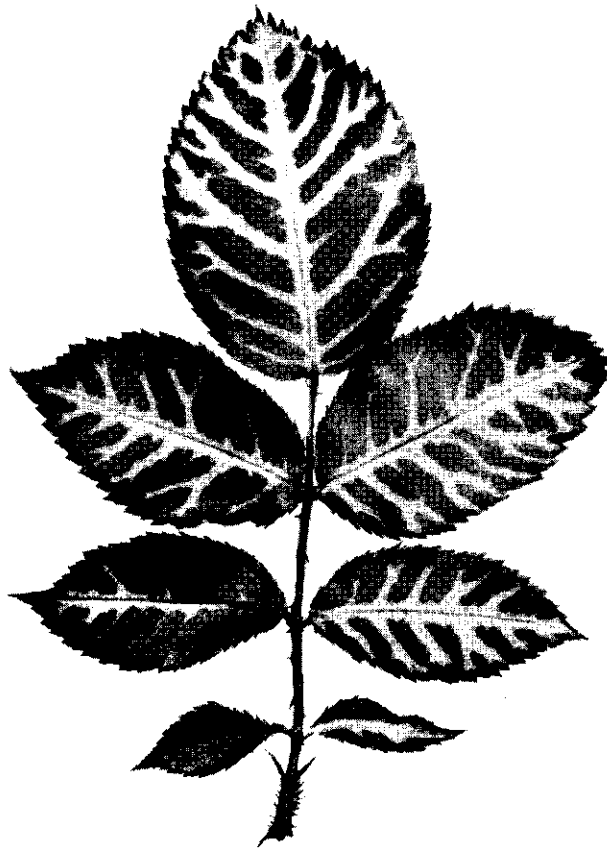


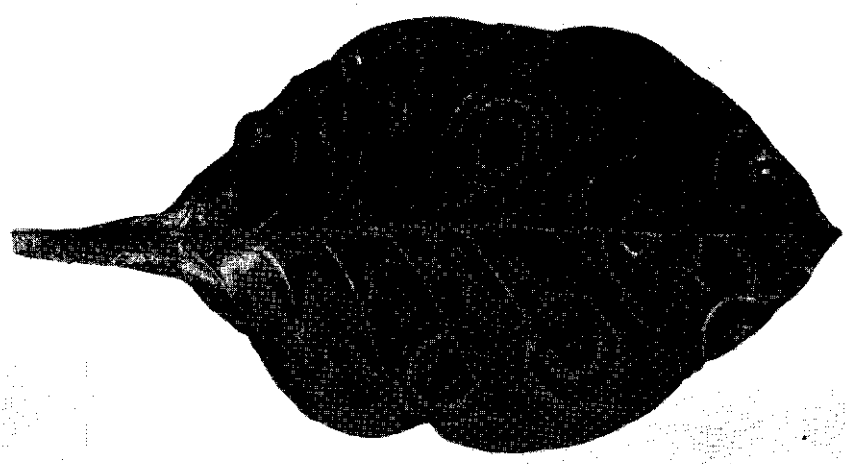
Fig. 10. Vein banding in rose leaf caused by rose mosaic virus. (Photo courtesy Plant Protection Service, Wageningen).

the light-coloured parts are grouped along the main veins e.g. in the 'red clover vein mosaic' (HAGEDORN *et al.*, 1959) and in apple mosaic (fig. 9). The phenomenon may sometimes give the impression of a vein chlorosis or vein yellowing, but the term mosaic indicates that the light-coloured areas are irregular and

that the discolouration is irregularly distributed over the venation of a leaf. Moreover, irregularly bordered adjacent tissue may be included. The term *vein banding* is used for those types of mosaic in which a rather regular range of light-, or sometimes dark-coloured tissue occurs along the main veins, e.g. 'raspberry vein banding' and rose mosaic (fig. 10). If the mosaic patterns occur mainly between the bigger veins, the name *interveinal mosaic* is often used, e.g. for symptoms induced by some strains of potato virus X.

If the discoloured parts are more or less round, the variegation is usually not designated as a mosaic, but as *flecking*, *spotting*, *speckling*, *specking*, *stippling* and *dotting*. These terms are almost synonymous except that there is some difference in size; the last four words are generally used for very small discoloured parts of the leaf. This group of more or less round lesions often occur on the site of inoculation. They may also consist of a light-coloured ring surrounding a normally green centre. Even concentric rings

Fig. 11. Unusual case of chlorotic concentric ringspotting in leaf of White Burley tobacco after systemic infection by a virus isolated from Eckelrader-diseased cherry (presumably raspberry ringspot virus). (Photo courtesy Miss H. J. Pfaeltzer, Wageningen).

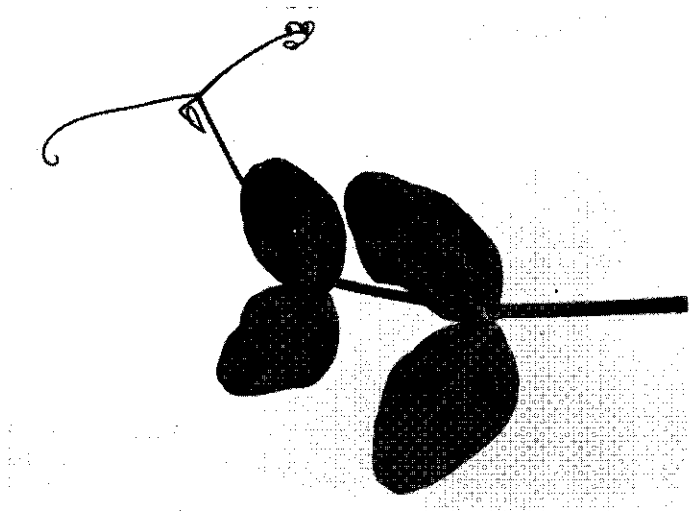


of dark- and light-coloured tissue may occur. Both types of spots are named chlorotic ringspots *(ringspotting)* (fig. 11). An important group of viruses is characterized by this type of symptoms in certain selected hosts such as the so called ringspot viruses. In ringspots the discolouration of tissue is often accompanied by necrosis. Sometimes the chlorotic or yellow spots are star-shaped, when the discolouration extends along some tiny veins: *asteroid spotting*. Light-coloured areas may also tend to become elongated, as in monocotyledonous plants, which have parellel-veined leaves. Then the variegation is designated as a *streaking* or *striping* (cf. also p. 39).

Flecks or their surrounding tissue are sometimes somewhat translucent, giving the impression as if they were 'water-soaked'. Then they may be named *oil flecks*. This flecking is characteristic of the initial stages of the Eckelrader disease or Pfeffinger Krankheit of sweet cherries (MULDER, 1951).

If the round discoloured areas have diffuse boundaries, the symptom is often called *mottling*. It differs especially from mosaic

Fig. 12. Mottling in pea leaves caused by the white clover mosaic virus. (After Bos *et al., 1959).*



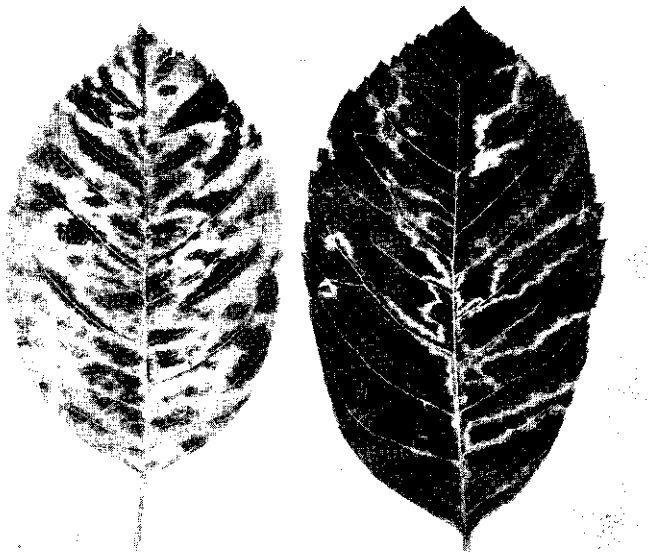


Fig. 13. Line pattern in juneberry (Amelanchier) caused by the pear ringspot virus. (Photo courtesy Plant Protection Service, Wageningen).

by the 'cloudiness' of the markings (fig. 12). In the literature there is quite some confusion as to the difference between mosaic and mottle. In some publications they are even used as equivalents. To the opinion of many virologists and of the present author, however, there exists a definite distinction, although it should be kept in mind that there may be intermediate forms of variegaton. For example a mosaic tending towards becoming diffuse easily gives the impression of a mottle. Mottling is a very common symptom such as in several stone fruit virus diseases and in some strawberry virus diseases. It is oftentimes used to designate virus diseases, such as 'strawberry mottle' (MELLOR & FITZPATRICK, 1960).

Sometimes the variegation consists of a conspicuous and often brilliant green-yellow pattern formed by single or multiple irregular lines or bands (fig. 13). This is called *line pattern*. The lines and bands may also occur as an *oak-leaf pattern* (fig. 14). The pattern may also consist of more or less irregularly shaped

Fig. 14. Oak-leaf type of line pattern in peach. (Photo courtesy Plant Protection Service, Wageningen).

rings. For this phenomenon the term *ring formation* is descriptive. The rings are often necrotic, however. A characteristic line pattern is produced in plum, peach and cherries by the 'line pattern virus' (e.g. CATION *et al.*, 1951).

In the light-coloured parts of the leaf, growth is often impeded, leading to deformation of the leaf (cf. secondary malformations, p. 84).

A peculiar type of discolouration is involved in the 'grey' disease or stripe disease of *Narcissus* (CALDWELL & JAMES, 1938). The silver-grey colouring of the leaves is due to an abnormal number of intercellular spaces.

Instead of being lighter than normal, the green colour of the leaves sometimes can be more intense than normal, e.g. in phony-disease of peach, where the green colour of the foliage is of striking depth and richness (HUTCHINS, 1933). A similar phenomenon occurs in 'wallaby ear' disease in Australia and in the presumably related 'nanismo ruvido' of maize in Italy (GRANCINI, 1958).

Fig. 15. Browning, necrotic streaking of stems and petioles, veinal necrosis, withering of leaflets, and purplish-brown necrosis of pods in pea, var. Rondo, naturally infected with pea 'early browning' virus. (After BOS & VAN DER WANT, *1962).* ▶


M.P.d.S.

Entirely different from the chlorophyll disorders described above are *reddening* and *purpling* due to the abnormal *anthocyanin formation*. These deviations are often quite similar to those induced by mineral deficiencies. A reddish-purple pigmentation, especially along the edges of the leaves, is characteristic of infection of aster yellows virus in clovers (HALISKY *et al.*, 1958) and in potato plants ('potato purple-top-wilt', RAYMER & MILBRATH, 1960). The barley yellow dwarf virus causes an intense orange-red colouration in oats: 'oat red leaf' ('Blattröte oder Rotblättrigkeit'; RADEMACHER & SCHWARZ, 1958). A striking brilliant reddening of the foliage of chokecherry is typical of the Western X-disease in the U.S.A. (REEVES *et al.*, 1951).

The production of dark-coloured melanin-like substances in dying tissues leads to *browning* and *blackening*. Naturally this colour change is very common in virus diseases (cf. especially the section on necrosis). The symptom of browning is typically represented by pea 'early browning' (BOS & VAN DER WANT, 1962) (fig. 15). The browning and blackening is often also involved in the production of ringspots. If death is caused by a rapid desiccation, the production of melanins is prevented, and the dry tissue may have a silvery-grey or whitish colour.

Both necrosis and the associated blackening, and desiccation and the associated greyish discolouration play a role in two peculiar phenomena called bronzing and etching.

Bronzing only seldom occurs and is due to a necrosis and collapse of epidermal cells overlying the still turgid, green and apparently healthy mesophyll tissue. It is caused by tomato spotted-wilt virus and occurs in circular markings, as a network following the finer veins, or almost continuously over larger areas of the leaves of tomatoes. The bronzing sometimes leads to a more general necrosis (SAMUEL *et al.*, 1930).

Not restricted to the epidermis, but still rather superficial is the symptom of *etching*. Here a shallow necrosis or collapse of tissue due to local desiccation gives the impression of corrosion, such as in 'tobacco etch disease' (JOHNSON, 1930). The etching also often proceeds to a more general necrosis (cf. also p. 50 and p. 52).

b. Colour deviations in stems

The stems can show chlorophyll disorders similar to those found on leaves, since herbaceous stems have similar chlorophyll-containing tissues as leaves. In peach calico e.g. even the twigs become creamy white in streaks (BLODGETT, 1944). Because of the comparatively small size of the stems, deviations in colour in the stems attract less attention than those in the leaves; hence they seldom give rise to names of diseases.

In 'potato stem mottle', caused by the tobacco rattle virus, the name is misleading since there are no mottle symptoms in the stem. The name 'stem mottle' had been given by Dutch field inspectors because of the fact that usually only one or two stems of a plant show symptoms (ROZENDAAL & VAN DER WANT, 1948). Thus the name refers to the irregular distribution of symptoms in the plants instead of to symptoms as such.

Most colour deviations in stems are due to necrosis. They will be described in detail in the section on necrosis. Since necrosis in the stem often finds its origin in the vascular system, the browning and blackening often occur as a *streaking* (cf. also p. 39 and p. 42) such as in several 'streak' diseases of peas, e.g. 'early browning' (BOS & VAN DER WANT, 1962) (fig. 15). Also whole stems may become black as a result of necrosis, such as in 'black root' of bean due to infection of common bean mosaic virus (JENKINS, 1941; GROGAN & WALKER, 1948).

In those cases where necrosis only occurs internally it may be observable externally as a greyish discolouration, such as in *Phaseolus vulgaris* infected with the white clover mosaic virus (BOS *et al.*, 1959).

c. Colour deviations in flowers

Colour deviations in the flowers form an important and interesting group of abnormalities. Even in old times they attracted attention, and they belong to the oldest-known virus symptoms. Tulips with a *breaking of the flower colours* are well-known from old Dutch paintings since 1619. They were described for the first time in 1576 by CLUSIUS. It is interesting to know that the attractive colours even led to a notorious traffic in tulip bulbs during the years 1634–1637, when fortunes were paid for a single nicely broken tulip.

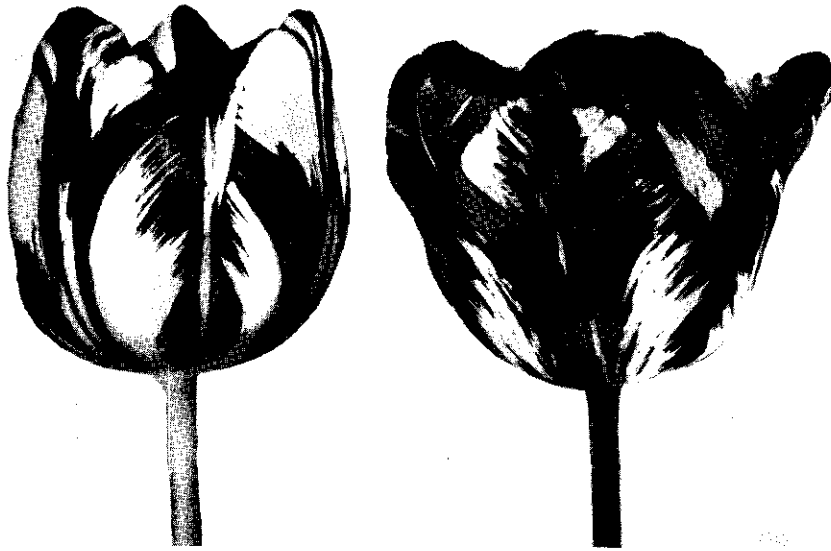


Fig. 16. Breaking of flower colours in tulip caused by tulip mosaic virus. Left, light breaking; right, light and dark breaking. (Photo courtesy Laboratory of Flowerbulb Research, Lisse).

The colour breaking is due to a local deletion of pigments from or to a local intensification or accumulation of pigments in the epidermal layer of the petals. In the first case, the white or yellow colour of the underlying mesophyll becomes visible (fig. 16, left), *light breaking*. It may give rise to very attractive colour patterns (cf. the many colour pictures of VAN SLOGTEREN & DE BRUYN OUBOTER, 1941). In the case of pigment intensification, *dark breaking*, small dark streaks or elongated flecks are developed. Both forms often occur together (fig. 16, right). These phenomena cannot be observed in white and yellow varieties since pigments are lacking in the epidermis of their petals. The disease is due to *Tulipa virus* 1. Some types of light breaking may be of genetic origin. Dark breaking can also be caused by rattle virus (VAN SLOGTEREN JR., 1958).

Breaking of the flower colours is quite common and well-known in several other plant species, e.g. in gladiolus after in-

fection with the bean yellow mosaic virus or with the cucumber mosaic virus (KLINKOWSKI, 1956) and in stocks *(Matthiola incana)* and common wallflower *(Cheiranthus cheiri)* after infection with the cabbage black ringspot virus (turnip mosaic virus).

Besides these variegation-type colour changes in flowers, general colour deviations also occur. The flower colours may be weakened, intensified or entirely changed. After infection with the chrysanthemum strain of cucumber mosaic virus chrysanthemum flowers with red, bronze or brown colour may turn entirely or partly yellow. Violet-red, light-red or pink flowers may become white-spotted or entirely white. In this case also, yellow flowers seldom change and white flowers never do (NOORDAM, 1952).

Greening or *virescence* (L. virescere = to grow green) also

Fig. 17. Wilting in gherkins proceeding from the top of a stem downwards caused by cucumber mosaic virus. (After TJALLINGII, *1952).*

belongs to the category of general deviations in colour. Instead of being normally coloured, the petals are more or less green as a consequence of chlorophyll development. Usually this phenomenon occurs together with deviations in form. Then virescence is a first stage in the complex of phenomena of antholysis (fig. 29–34) (cf. the section on malformations).

d. Colour deviations in fruits and seeds

Fruits also may show deviations in colour. Usually these are due to chlorophyll disorders, similar to those occurring in leaves and stems, or to necrosis. As beauty defects they influence the market value and may be of great economic importance. In plants with big fruits, such as in gherkin (pickling cucumber) infected with the cucumber mosaic virus (e.g. TJALLINGII, 1952), colour changes attract special attention. This mosaic is also associated with deformations of the fruit (cf. p. 84). Several viruses, such as tobacco mosaic virus and tomato spotted wilt virus, may induce colour deviations in tomato fruits (e.g. DOOLITTLE, 1951). Colour changes in fruits due to necrosis will be mentioned on p. 57.

In Japan a brown specking or brown mottling of soybean seeds was reported to be a disorder due to infection by the seed-borne soybean mosaic virus (KOSHIMIZU & IIZUKA, 1957).

IV. Water deficiency

In plant diseases host tissues often show a lack of water: *water deficiency* or *water shortage*. This brings about a loss of turgidity, leading to *wilting*, or a total loss of water or *desiccaton*, usually leading to *withering*. Withering means desiccation associated with shrinking and shriveling. In some languages (see index) there are no differential terms for desiccation and withering. In case of wilting the tissue shows a flaccidity and it may recover. Wilting may also proceed to withering which is irreversible.

A very striking wilting occurs in gherkin (pickling cucumbers) as a result of infection with cucumber mosaic virus when the second week after infection is characterized by cloudy and cool weather (maximum day temperature 20° C or lower) (TJALLINGII, 1952) (fig. 17). Withering is very common in virus diseases. In peas with early browning withering of leaflets occurs as a consequence of necrosis of veins and petioles (fig. 15).

Desiccation may be confined to certain parts of the leaf. In *Phaseolus vulgaris* infected with tobacco necrosis virus ('bean stipple streak') the leaves may show necrosis of the tiny veins in restricted areas of the lamina. Desiccation of the associated interveinal tissue results. Since the dry area is surrounded by turgid tissue, the loss of turgescence is hardly noticed, and no shriveling occurs. Local lesions very often consist of desiccated tissue and then have a grey or whitish colour.

A peculiar type of local desiccation is represented by *etching*. Here a collapse of superficial tissue gives the impression of corrosion. This local collapse may be due to desiccation as well as to necrosis (cf. also p. 45 and p. 52).

As yet very little is known as to the exact origin of water deficiency due to virus infection. Obviously it is induced by a reduced supply of water and/or to an excess of transpiration. The reduced supply may be due to necrosis in the vascular bundles, to a deposition of gum in the vessels and other xylem cells, or to excessive development of tyloses in the wood. The latter possibility has been described for grapevine infected with Pierce's disease virus (ESAU, 1948b). In this grape disease it leads to a sudden wilting of vigorously growing young vines and the drying of leaves. In gherkins showing the above-mentioned wilting, TJALLINGII (personal communication) could not find any disturbance in the vascular system. Moreover, in this disease, the wilting starts in the tops of the stems and proceeds downwards. Both facts suggest an excessive transpiration.

V. Necrosis

The dying of cells or tissues is called *necrosis* (Gr. nekroun = to make dead, to mortify) and is a quite common symptom in virus diseases. It usually takes place very rapidly, and a clear-cut borderline between dead and living tissue can usually be observed. Necrosis is made especially conspicuous by the production of dark-coloured melanin-like substances, as has been described in the section on colour deviations. Since location and type of necrosis is often characteristic, this symptom may be of diagnostic value. It may affect superficial cells, or it may occur in deeper layers of tissue. It may involve several different tissues or be restricted to one type of tissue.

Necrosis frequently develops at the site of virus entry. Then it is often confined to the inoculated cell together with some surrounding cells, giving rise to a necrotic local lesion. The mechanism of this severe local reaction is not understood. Such a *hypersensitivity* often leads to a limitation of the infection, preventing the plant from being infected systemically.

This hypersensitivity can be of practical importance as it may result in resistance to the given virus under natural conditions. A good example is the field resistance to the common bean mosaic virus (*Phaseolus virus* 1) of bean varieties descending from the North American Corbett Refugee (QUANTZ, 1957, 1958).

Under humid conditions necrosis is often succeeded by *rotting*. This secondary phenomenon, however, is due to secondary fungi or, more often, to bacteria. They decay the dead material saprophytically (cf. secondary phenomena). Under dry conditions the necrotic tissue may dry out. Rotting is never caused by viruses, since virus activity is associated with living tissue only. In lasting necrotic lesions, such as in woody stems, this secondary rotting may give an ulcer-like effect, as often occurs in cankers (p. 55).

a. Necrosis in leaves

Necrotic spotting or *necrotic speck(l)ing* in the interveinal tissue may occur as a result of local virus infection e.g. on leaves of *Nicotiana glutinosa* or *Phaseolus vulgaris* after mechanical inoculation with tobacco mosaic virus or any of a number of other viruses. Often these spots show (concentric) rings consisting of necrotic, yellow and dry tissue: *necrotic ringspotting* (fig. 2). If the deviation only consists of a pattern of necrotic ring- and arc-like figures it can be termed *necrotic ring formation.*

Sometimes the necrotic spots may be due to a systemic reaction. It may be that this is due to the movement of relatively few infectious particles resulting in a restricted number of places where infectious units become established (fig. 2). A good example of systemic necrotic spots is 'necrotic stipple' in store cabbages, a symptom caused by the cauliflower mosaic virus (VAN HOOF, 1952).

In those cases where the necrosis is only superficial and generally less localized, it gives the impression of a corrosive

effect and is often called *etching* (cf. p. 45). This superficial effect, however, may also be due to a shallow desiccation of tissue. In case of *bronzing* the necrosis and collapse is restricted to epidermal cells overlying the still turgid and green mesophyll tissue (p. 45).

When large numbers of necrotic lesions develop upon mechanical inoculation they may coalesce and form dead areas. The necrotic lesions themselves sometimes also may gradually or rapidly enlarge, producing a more systemic necrosis such as in a number of streak diseases of peas. In these diseases usually the distribution of necrosis is irregular, but entire leaves may eventually die. After reaching the veins and spreading rather quickly within the vascular system of the plant, these viruses may induce a *veinal necrosis* in systemically infected leaves. After some time the necrosis may proceed to the interveinal tissue or desiccation of this tissue may result, e.g. in pea plants with early browning (fig. 15).

Necrosis of the veins is caused also by tobacco necrosis virus in bean leaves: 'bean stipple streak'. However, it is confined to a restricted area around the point of inoculation.

b. Necrosis in stems

After reaching the veins, the necrosis generally does not remain restricted to the leaf but continues along the petioles to the vascular system of the stem and afterwards from the stem to the higher leaves. This necrosis often leads to a disturbed water supply and consequently to wilting and withering of the leaves concerned. This is striking in pea plants infected with early-browning virus (fig. 15).

An interesting type of vascular necrosis is 'black root' (JENKINS, 1941) in a number of snap-bean varieties having field resistance to the common bean mosaic virus (cf. p. 51). In these varieties the virus remains confined to small necrotic spots. At temperatures above 20° C, however, the virus is able to become systemic and to induce necrosis in vascular tissues of all plant parts such as roots, stems and pods. The same systemic vascular necrosis occurs in these hypersensitive varieties at normal temperatures if the virus is introduced into the vascular system by grafting (GROGAN & WALKER, 1948).

Quite commonly a systemic reaction leads to death of young sprouts or tops of stems, e.g. in beans after infection with a special strain of the bean yellow mosaic virus or in some potato varieties after infection with potato virus A or virus X. This *top necrosis* is sometimes called 'acro necrosis'.

Different possibilities exist concerning the origin and location of necrosis in the anatomy of stems.

In diseases caused by viruses that are more or less limited to the phloem, the necrosis generally is restricted to the phloem. A classical example of this is *phloem necrosis* in potato plants infected by leaf-roll virus (QUANJER, 1913). It involves the sieve tubes and companion cells. This necrosis can be observed by means of a microscope only. In this potato disease phloem necrosis sometimes proceeds into the tubers and causes 'net necrosis' (p. 55). Two other examples of phloem necrosis are sugar beets with curly top (ESAU, 1933), and Gramineae affected by the barley yellow dwarf virus (ESAU, 1957). Phloem necrosis is of special importance in such diseases as citrus tristeza, where it occurs immediately below the bud union in sour orange rootstocks, grafted with infected sweet orange scions (SCHNEIDER, 1959). This necrosis leads to depletion of starch in the roots and subsequent rotting of these roots. As a consequence the aboveground tree shows *decline*, consisting of a progressive starvation, wilting, defoliation, and associated phenomena (cf. p. 23).

Because of the transport of virus through the phloem many types of necrosis are initiated in the phloem of the vascular bundles, but extend eventually to other tissues. In the abovementioned black-root disease of beans, necrosis affects not only the phloem but also the cambium and the outermost layer of xylem (JENKINS, 1941).

In the 'streak diseases' of potato, grouped under the name 'acro necroses' by QUANJER (1931), necrosis arises in the phloem and then spreads into the neighbouring tissues in all directions, most markedly towards the xylem. The types of internal necrosis are often manifest to the naked eye as a diffuse dark-coloured *streaking* on stems, petioles, and main veins.

Stem necrosis also may find its origin in the parenchyma. In stems, in petioles, and main veins of French beans infected with the white clover mosaic virus, parenchyma cells in the pericam-

bium (tissue between phloem and cortex) or between the xylem elements, or groups of interfascicular parenchyma cells may be necrotic (Bos, unpublished data). This necrosis is preceded or accompanied by a deposition of gum (cf. p. 28) and is observable externally as dark greyish streak-like discolourations.

In potato plants affected by potato virus Y ('leaf drop streak', 'stipple streak' or 'acropetal necrosis'), necrosis occurs in the collenchyma of the aerial organs and sometimes extends to other tissues of the cortex but not to the vascular bundles. In the petioles even the parenchyma between the bundles may become affected (QUANJER, 1931). The necrotic streaks are visible from the outside.

In tobacco stems infected with tobacco rattle virus, the pith and the cortex show considerable necrosis (BÖNING, 1931).

In addition to these internal necroses, a stem can have more superficial necrotic symptoms, restricted more or less to the cortex. Presumably this necrosis resembles the necrotic lesions in leaves and petioles. For example, potato stem mottle, caused by the tobacco rattle virus, shows a superficial necrosis, starting in the leaf and proceeding to the cortex of veins, petioles and stems without affecting the vascular bundles (QUANJER, 1931).

In the literature the term 'streak' is often used for these necrotic stripes. This is very confusing, however, for it is inadequate. Streak literally means 'stripe' and may be used for a stripe-shaped discolouration as well as for a stripe-shaped necrosis. In cereals yellow flecks are elongated because of the parallel venation of the leaf. This led to the name 'cocksfoot streak' for a mosaic disease of cocksfoot, where no necrosis occurs (cf. p. 39).

A necrosis in the bark of elm trees has been described as a virus disease under the name 'elm zonate canker' (SWINGLE & BRETZ, 1950). The symptoms appear in the bark as concentric rings of dead and living tissue in the cortical or phloem tissue. Later the areas enlarge and the necrosis may extend into the xylem. The necrosis may cause the bark to split. Frequently stems and branches are girdled, after which their upper portions die. For this localized necrosis in stems and twigs leading to death of the bark up to the wood, in the English language the term *canker* is commonly used. The name *bark necrosis* might be clearer. According to a discussion of literature by ZYCHA (1955) no accurate definition of the term canker is known. The localized

necrosis may induce, especially in the so-called 'perennial cankers', the production of a tumorous callus around the wound. Then the phenomenon has many features in common with cankerous tumours (cf. p. 65) and may be called *tumorous canker*. (One should keep in mind that the term canker is not equivalent to the Dutch term 'kanker' and the German 'Krebs'. This will be discussed further on p. 65).

In plant pathology for cankers sometimes the name 'bark anthracnose' (Gr. anthrax = coal, nosos = disease) is being used. Anthracnose, however, is the name for a disease. The disease is characterized by ulcer-like lesions.

Tubers of potatoes may show a series of necrotic phenomena. In spraing or corky ringspot, presumably caused by a virus related to potato stem mottle (tobacco rattle), the cut surface of a tuber shows arc- or ring-like necrotic patterns; it is a type of necrotic *ring formation* (for a survey of literature cf. EIBNER, 1959, and WALKINSHAW & LARSON, 1959). Since the symptom is accompanied by some cork formation, this led to the name 'corky ringspot' for the disease. The Dutch term 'kringerigheid' points to the irregularity of the rings. It is difficult to translate this term into English. The German name 'Pfropfenbildung' (formation of wads or pellets) expresses the three-dimensional nature of the symptom. These spatial structures have a similar origin as the two-dimensional necrotic rings in leaves.

In corky ringspot LIHNELL (1958) distinguishes between primary and secondary symptoms as regards their localization in the tuber. The primary symptoms often have the appearance of emanating from a centre just at the periphery of the tuber. The secondary symptoms, on the other hand, are mostly restricted to the heel end and are often arranged around the hilum of the tuber as a centre. The difference can be explained by assuming the incitant to enter from the soil through the skin in the first case and to enter the tuber systemically through the hilum in the latter case.

The tubers of a number of potato varieties, especially in North America, produce 'net necrosis' after infection with leaf-roll virus. In the subsurface tissue of the tuber dark brown flecks, stripes and reticulated figures develop, which are composed of necrotic sieve tubes and companion cells (fig. 18) (FOLSOM *et al.*, 1938). It is nothing but *phloem necrosis* (cf. p. 53). Because of the irregular distribution of the vascular bundles in

Fig. 18. Phloem necrosis in tuber of Green Mountain potato caused by potato leaf-roll virus. (After FOLSOM *et al., 1938).*

the tuber, the cut surface of a tuber shows net-like markings. This necrosis is plainly visible to the naked eye. The so-called 'tuber blotching' (or sometimes 'pseudo net necrosis') usually occurring in the parenchyma cells of both cortex and pith of potato tubers, is caused by the potato aucuba virus. The necrosis is easily visible as rusty to dark brown spots and stipples within and outside the vascular ring (CLINCH *et al.*, 1936). Tuber blotching is the three-dimensional equivalent of necrotic *spotting* in the leaf.

The phenomena in potato tubers mentioned above, differ from the so called 'tuber rust spot', the German 'Eisenfleckigkeit'. These spots are more diffuse and are assumed to be of physiological nature (EIBNER, 1959).

Since the terms 'corky ring spot', 'net necrosis' and 'tuber blotching' are used to indicate specific potato diseases, and since the

deviations concerned can be derived from other single symptoms, they should not be applied in general as names of symptoms.

c. Necrosis in fruits

Necrosis may also occur in fruits. Pods of beans with stipple-streak virus (tobacco necrosis virus) and pods of peas with early-browning virus (fig. 15) often show necrotic ring patterns. Another exemple of necrotic rings in fruits is that in tomatoes caused by tomato spotted wilt (KOVACEVSKI, 1959).

In pear fruits infected with stony-pit virus, necrotic centres occur in addition to concentrations of sclerenchyma cells (KIENHOLZ, 1939).

VI. Cork formation

The formation of cork is a normal phenomenon in plants. It quite commonly occurs in diseased plants and often is a secondary phenomenon due to wounding. *Cork formation* also is often involved in virus diseases. In contrast to the previous symptom groups no abnormal cells are produced; instead normal cells are incited to divide and form cork cells. This deviation is more or less an organizational disturbance. Cork formation in virus-infected plants has very seldom been the subject of serious anatomical research, hence little accurate information is available.

KLINKENBERG (1940) made some investigations on the anatomy of abnormal cork in the roots of *Lupinus polyphyllus* affected by 'sore shin', in her material presumably caused by cucumber mosaic virus. The cork occurs in a continuous layer at the bases of small intumescences on the roots and also in deeper layers around intercellular spaces filled with gum or around bigger groups of necrotic cells.

In 'psorosis' of citrus, development of cork in the bark of stems cuts off the outside layers, which die and form the dry scales or flakes of bark (FAWCETT & BITANCOURT, 1943). This *bark scaling* is indicated by the name psorosis, which means a disease characterized by psora or scab.

A peculiar and conspicuous type of cork formation develops on the fruits of apple infected with 'apple rough skin' virus. This *rough skin* phenomenon results in rough corky brown

patches on the skin of apple fruits. The patches may be small and somewhat circular but may also occur in rings or elongated stripes, whilst the fruits of severely infected trees often show a roughening of large parts of the skin. Sometimes the rough patches are cracked, and the fruits may show a slight deformation that is due to local growth retardation (VAN KATWIJK, 1955, 1956). *Star cracking* occurs in the corky patches in a presumably similar or at least related disease. This phenomenon gave rise to the name 'apple star cracking virus' (JENKINS & STOREY, 1955).

VII. Malformations

The symptoms characterized by growth reduction, colour changes, water deficiency and necrosis found their origin in visible effects on the cell. In a large group of virus diseases, however, the cells themselves may appear entirely normal, but the so-called correlation or mutual relation of cells, tissues, and even organs during development may be abnormal. This abnormal growth or development leads to *malformations* or deviations in the structure and form of plant parts or entire plants.

The group of malformations is a rather complicated one. Cytological abnormalities are sometimes also involved, hence the distinction between malformations and the previous groups of symptoms is not always clear-cut.

Malformations may be primary or secondary. In the first case the aberrations are among the first visible symptoms of infection and are directly caused by infection. Secondary malformations are not evident until after the plant develops certain symptoms such as localized yellowing or necrosis, which may lead to malformation of the organ concerned. They are indirectly due to virus infection. The distinction between these types of malformations, which is not always sharp, will be discussed more in detail in the proper parts of this chapter.

A. The *primary malformations* are among the first visible deviations due to the virus infection, and presumably are due to a disturbed action of phytohormones (cf. also p. 67), resulting from a disturbed transport or distribution of hormones or from a decrease or increase in hormone level. Since the level required for optimal growth differs for different plant parts, a shifted

hormone level also may disturb developmental proportions. VON DENFFER (1952) has already ascribed the proper sequence in leaf forms in the normal plant development to changes in hormone level.

Malformations produced in this way can be divided into two groups, in the same way that KÜSTER (1911, 1925) did with the plant gall abnormalities produced by animals and fungi. The *histoid* or *histological deviations* (histoid = tissue-like) are due to an abnormal organization of distinct tissues or of tissues within a distinct organ. In *organoid* or *morphological deviations* (organoid = organ-like) the tissues and organs involved may be normal, but the organization of the organs, i.e. the relation between the organs, is abnormal. The adjectives histoid and organoid indicate that the abnormal products are tissue-like and organ-like. One should keep in mind that there exists no clear-cut borderline between the histoid and the organoid deviations. Cytological abnormalities may be involved, especially in histoid aberrations; these then also may result from improper hormonal balance.

A term that is often used in connection with these growth abnormalities and that needs explanation is the name *proliferation* (L. proles = offspring or sprout; fero = to bear). The term refers to uncontrolled 'yeast-like' sprouting of cells (cf. also hyperplasia), but is also used for uncontrolled growth and development of tissues and organs (cf. also p. 27). Especially in other languages (Dutch: 'woekering'; German: 'Wucherung') it also has a connotation of abnormal development at the cost of surrounding cells, tissues and organs.

A 1. Since normal organs are not produced, the *histoid malformations* cannot be described in the terms used in plant morphology. Most of them are due to hyperplasia.

Histoid *enations* (L. e = from; nasci = to be born, to come into existence, to grow or develop) are peculiar protruding growths, being rather restricted in size. They occur in lucerne, mostly only one per leaflet, after infection with a virus found in Rumania (BLATTNY, 1959). A presumably related graft-transmissible disease has been observed in white clover in Italy and is being studied by GRANCINI and the present author (fig. 19). In these diseases, the enations are usually restricted to the main

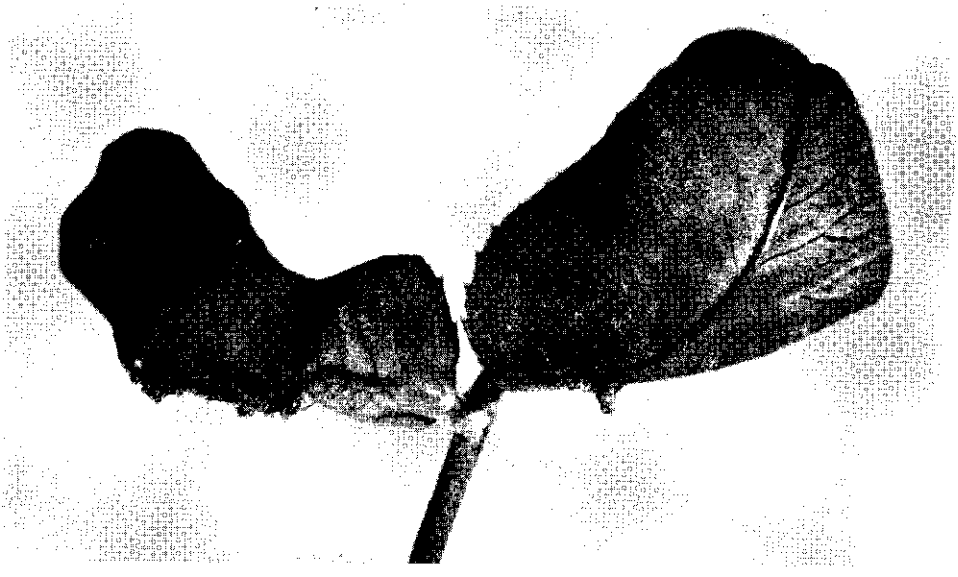
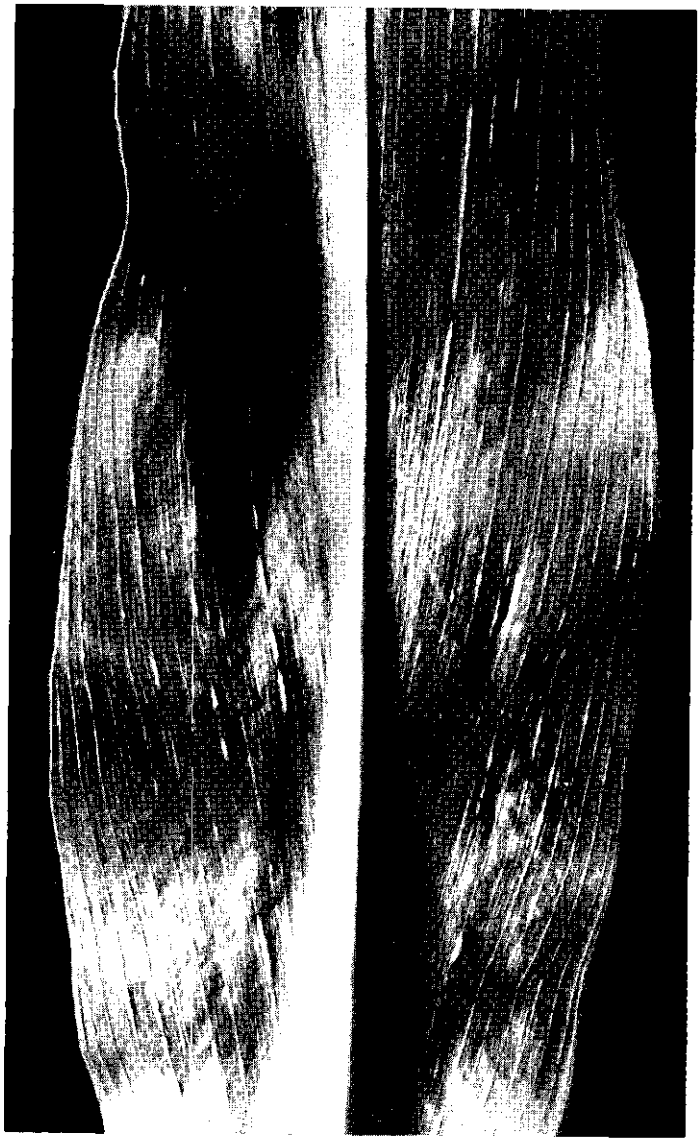


Fig. 19. Histoid enations in white clover after infection with an unidentified graft-transmissible virus.

and side veins. They are the more conspicuous since they are located on top of a conical or funnel-shaped protrusion on the underside of the leaflet. The excrescences may vary in form from large or small papillae to short whitish spines. Other interesting histoid enations are those on the veins of citrus leaves as a result of infection with the 'citrus vein-enation' virus (WALLACE & DRAKE, 1959).

Also belonging to the category of enations are the elongated ridges of tissue or swellings on leaves e.g. produced by the narcissus mosaic virus, the Fiji-disease virus of sugar cane, and the dwarf-disease virus of maize. The small ridges of tissue or swellings on the upper surface of the leaf of stripe-diseased narcissus (narcissus mosaic virus) are initiated in the palisade cells and are due to hypertrophy (enlargement) and hyperplasia

Fig. 20. Elongated histoid enations on the veins of the under surface of a maize leaf caused by a rough-dwarf disease virus. (After GRANCINI, *1958).* ▶



(increase in number) of palisade cells. Eventually, the proliferating tissue protrudes through a crack in the epidermis. This proliferation occurs simultaneously in a narrow strip of palisade tissue, thus forming a ridge instead of a rounded projection (CALDWELL & JAMES, 1938). These swellings to some extent resemble the so-called intumescences, caused in many plant species by excessive humidity. The small elongated excrescences on the under surface of the leaves of sugar cane, infected with Fiji-disease virus, result from abnormal proliferation of the phloem or of tissues immediately adjoining the phloem (KUNKEL, 1924). Thus they extend along the under surface of the veins. A similar phenomenon occurs in the rough-dwarf disease of maize (fig. 20) (GRANCINI, 1958). Here BIRAGHI (1952) demonstrated the underlying proliferation of phloem tissue.

Although there is no sharp-cut borderline, the so-called *tumours* (Am. *tumor*) generally are less restricted in size than the enations and then are due to a more irregular and less limited development or proliferation of cells and tissues. The tumours are amorphous growths without any reasonable organization. They have many features in common with human and animal cancers, hence they have attracted much attention. Of course there are some differences due to differences in the ontogeny of animal and plant tissues (BLACK, 1952). The origin and characters of tumours differ according to the virus, the host plant, and the specific part of the host concerned.

Tumorous swellings in leaves generally are rather restricted in size. The swellings on the lower surface of the veins of crimson clover ('clover big vein'), induced by the wound-tumour virus, are more or less enation-like. They arise as a consequence of the development of many small tumours in the phloem of the veins. In the leaf petiole, stem, and root, many of these tumours are only found internally, not showing on the surface as protuberances (LEE & BLACK, 1955).

Well-known and intensively studied are the tumours induced by the wound-tumour virus on stems and on the roots of sweet-clover *(Melilotus alba* and *M. officinalis)* (fig. 21) and on the roots of sorrel *(Rumex acetosa)* and a number of other plant species (BLACK, 1945). The stem tumours may attain a diameter of about 1 cm. They are produced in systemically infected plants only after wounding (BLACK, 1946). A natural wounding occurs

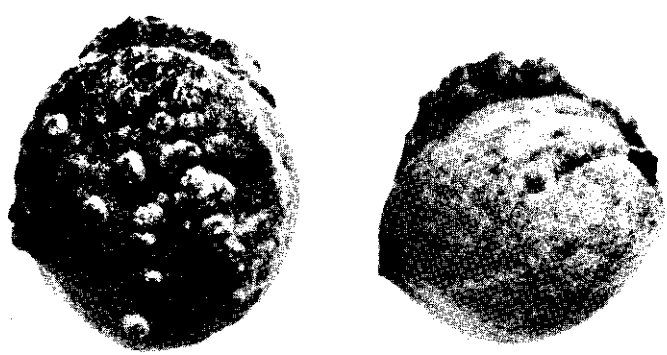
Fig. 21. Root and stem tumours in sweet clover (Melilotus officinalis clone C 11) caused by artificial infection with the 'wound-tumour' virus. (Photo courtesy Dr. L. M. Black, Urbana, Ill.).

in roots producing side roots, which are initiated in the pericycle and mechanically break through the cortex. Tumours develop close to the wounded cells in the pericycle (LEE, 1955) and may be formed even at the bases of bacterial nodules. Very interesting are the woody tumours on young rough lemon and West Indian lime trees, which have recently been associated with the above-mentioned citrus vein-enation virus (WALLACE & DRAKE, 1960).

Histoid outgrowths may even occur on fruits, such as in the peach 'wart' disease (fig. 22) (e.g. BLODGETT *et al.*, 1951). Bleached bumps or raised welts may develop on or near the stylar end of young fruits; these often involve half or more of the fruit. The wart-like structures are rather superficial, but the underlying tissue is coarse and filled with gum pockets. In some cases the warty tissue is very hard and bone-like, but usually it is tough and leathery.

Many virus-induced enations and tumours resemble swellings produced by parasitic organisms, such as insects, nematodes and bacteria. At present attention is concentrating on the resemblance of virus-induced enations and those produced by leafhopper saliva. For instance, ANTOINE (1959) and BAUDIN (1960) were able to distinguish 'pseudo Fiji' of sugar cane in Madagascar, induced as a toxic effect of leafhopper feeding, from the true virus-induced Fiji disease.

Fig. 22. Tumorous outgrowths on fruits of peach affected by 'peach wart' disease. (Photo courtesy Dr. J. A. Milbrath, Corvallis, Oregon).



The symptomatological resemblance of enations and tumours caused by viruses to swellings produced by parasitic organisms, indicated as galls, already led to the introduction of the term 'gall' into plant virology. An example is the name *Galla fijiensis* HOLMES for the Fiji disease of sugar cane. More recently the cecidologist TROTTER (1954) and the plant virologist BLATTNY (1961) used the name 'viro cecids'.

The name 'gall' must be considered inaccurate, however, for galls are swellings caused and inhabited by animal or plant parasites (KÜSTER, 1911, 1925). They are of critical ecological importance to the parasites. This does not hold for the malformations due to virus infection, because the virus occurs in the other parts of the plant as well. Moreover, we have no evidence that the tumour is necessary to the survival of the virus. The Dutch cecidologist DOCTERS VAN LEEUWEN (1959) agrees with the present author's opinion that growth abnormalities induced by viruses should not be classified as galls.

Also more extensive swellings may develop. In the stems of sweet cherry, var. Napoleon, in Oregon, U.S.A., cherry black 'canker' virus induces slightly swollen areas, that later split and grow into rough black 'cankers' (fig. 23) (ZELLER *et al.*, 1951). Prune diamond 'canker' (SMITH & THOMAS, 1951) is a somewhat similar virus disease. However, these swellings are primarily tumours. It should be kept in mind that the name canker especially refers to localized necrosis in the bark of woody stems (cf. p. 54). In these examples, necrosis presumably occurs secondarily, whereas in cankers necrosis occurs primarily, afterwards sometimes inducing the production of a callus around the wound, especially in the so-called 'perennial cankers'. There is a confusing linguistic problem connected with these phenomena. The Dutch term 'kanker' means both canker and cancer. The English term cancer (carcinoma) is only used in medical science for malignant tumorous, usually ulcerating swellings. The German 'Krebs', which is equivalent to cancer, however, is also used for cancerous deviations in plants. Therefore it is suggested, that these cancer-like abnormalities, clearly having necrosis, be called *cankerous tumours*. (If the callus formation occurs secondarily they should be called *tumorous cankers*, cf. p. 55).

Even entire stems or shoots may swell, resulting in a symptom termed *shoot swelling*. A good example is the notorious 'swollen shoot' disease of cocoa in Western Africa (POSNETTE, 1947). Suckers arising from the base of the trunk are especially likely to show pronounced swellings, causing them to have a diameter twice that of the normal stem. They may be nodal or internodal

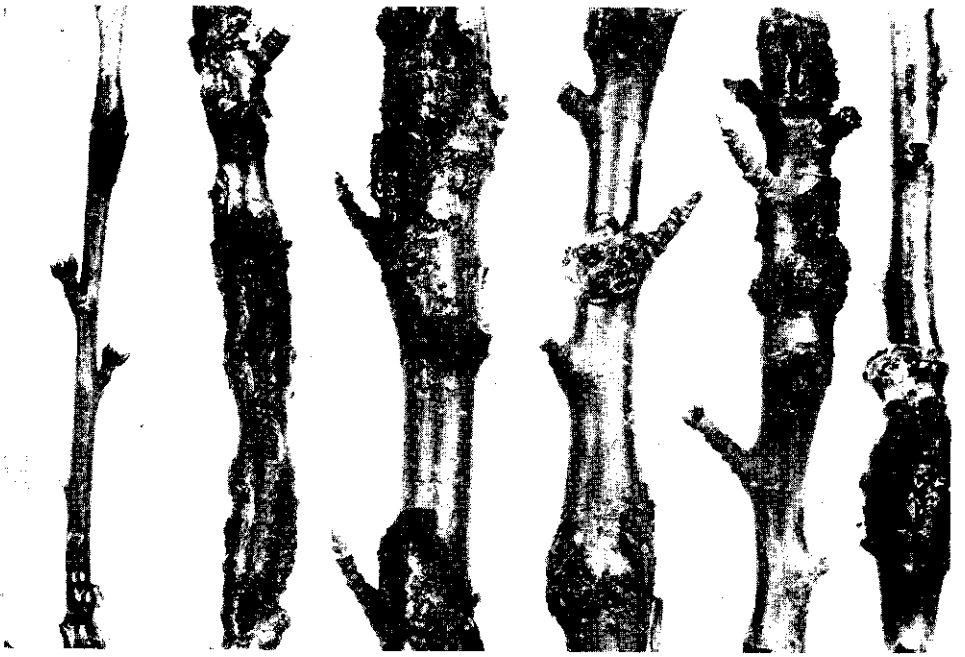


FIG. 23. *Cankerous tumours in twigs of Napoleon cherry affected with cherry 'black canker'. (After* ZELLER *et al., 1951).*

but are often terminal. The swelling is due to an increase in xylem tissues. Only slight proliferation of phloem tissue occurs.

A singular histoid modification caused by abnormal secondary growth is the *flattening of branches* in a virus disease of apple known as 'flat limb'. The first symptoms appear in two- or three-year-old limbs or branches. These are irregularly flattened (German: Flachästigkeit) or provided with broad ribs (German: Rillenkrankheit). With increasing thickness of the branch, the furrows become deeper. Because of one-sided growth reductions, distortions of branches may occur. Sometimes spindle-shaped swellings up to twice the normal diameter have been observed. Transverse sections show a highly reduced production of xylem in those parts of the section with a decreased radius. The cortex in the same regions, however, has more than twice the normal thickness (BLUMER, 1956).

A 2. Within the *organoid malformations* not only the cells but also the tissues and organs usually are normal. The organizational disturbances lead to deviations in the external structure of the plant, which can be described in morphological terms. These morphological aberrations, especially those of the flowers, have attracted the attention of many botanists for a very long time. They were studied formerly as curiosities, but later their morphological significance was considered in a special branch of botany named *teratology* (Gr. teras = wonder, monster) (cf. the textbooks of MASTERS, 1869, and PENZIG, 1921–1922).

At present we know that many teratological phenomena, sometimes named *teratomata* (and formerly less properly called 'monstrosities'), are symptoms of diseases, some of which are due to viruses. For a thorough discussion see BOS (1957b). A consideration of these abnormalities leads us into the field of pathological morphology or patho-morphology.

Apparently the organoid malformations are practically all due to hormonal disturbances, the hormones being the growth- and correlation-regulating substances. A number of malformations of leaves caused by viruses even may be quite similar to those due to an overdosing of hormonal weedkillers.

Deviations in the morphology of leaves are quite common in virus diseases. Although these developmental aberrations influence the gross habit of the leaves, it is often difficult to describe them in morphological terms. Most of them can be ascribed to unbalanced development of veinal and interveinal tissue.

Often the growth of the laminar tissue is greatly reduced, leading to *leaf narrowing* as in cherries with Pfeffinger or Eckelrader disease, where the deformation is accompanied by a deeper serration of the edge of the lamina than normal (fig. 24, middle row), or in yellow lupines with lupine mosaic. A very common example is tomato 'narrow-leaf' caused by infection with tobacco mosaic virus or with cucumber mosaic virus (fig. 24, above). Leaflets of infected tomatoes may have a fern-leaf appearance. The lamina may even be almost or entirely absent; sometimes only the main vein develops. This extreme form of leaf narrowing is called *shoe stringing* and sometimes 'lacing'. TEPFER & CHESSIN (1959), in a study of the development and anatomy of narrow-bladed and shoe-string leaves of Turkish

tobacco, Xanthi strain, after infection with tobacco mosaic virus, found that the shoe-string leaf in its extreme form is entirely radial in symmetry, with no vestige of a lamina. The effect of leaf narrowing in palmate leaves is shown by grape vines after infection with vine-roncet, fan-leaf, or court-noué viruses (fig. 24, below). The laminar tissue shows varying stages of reduction. The palmate leaves start with greater dentation or deeper lobation of the margins, and the five main veins of the leaf become gathered together toward the midrib, similar to those of a partially closed fan. This *fan-leaf formation* suggested the name 'fan-leaf' disease (HEWITT, 1950).

In contrast to the above, some leaf abnormalities result from reduced growth of the veinal tissue in comparison with the interveinal tissue. This leads to a lumpy, bubbled surface of the leaf such as in tobacco leaf curl, with sunken veinlets and elevated interveinal tissue. This is termed *rugosity*. For those instances where the deformation is more irregular, and is associated with furrowing and wrinkling of the lamina, the terms *crinkling* and *curling* are used, as in turnip 'crinkle' and sugar beet 'leaf curl'. These phenomena, especially the more irregular ones, however, may also occur as secondary malformations as a consequence of anatomical or other disorders of the veins (cf. p. 85). Obviously, it is hard to distinguish between crinkling and curling, although curling generally indicates a more irregular and sometimes distortive type of deformation.

Another possibility of imbalanced development is the reduced development of the petiole and midrib of the leaf or of the rachis of a compound leaf. This is typical for the potato bouquet disease in potato (KÖHLER, 1952). This phenomenon leads to crowding of the leaflets and sickle-shaped downwards curving of its midribs.

◀ *Fig. 24. Leaf narrowing. Top row, in tomato infected with tobacco mosaic virus; middle row, in cherry infected by Pfeffinger-disease virus (note the deeper dentation); bottom row, in grape infected with 'fan-leaf' virus and called fan-leaf formation; healthy leaves, left.* (Drawing Miss I. Zewald, Department of Plant Taxonomy and Plant Geography, Agric. Univ. Wageningen).

Epinasty (Gr. epi = on, upon; nastos = pressed close) is a quite common phenomenon in virus-infected plants, but may also be due to a number of other factors. It finds its origin in an excessive growth of the upper surface of the organ, such as the petiole and leaf blade. This leads to downwards curling of the entire leaf. It is not known whether the regular upwards curling, as in potato 'leaf roll', or downwards curling over the whole length of the leaf, both known as *leaf rolling*, is a comparable phenomenon.

A very peculiar morphological aberration of the leaf is the abnormal enlargement or *hypertrophy* of stipules of apple leaves, caused by the apple witches' broom virus (fig. 25). This symptom is of important diagnostic value.

Another deviation influencing the gross habit of the plant is premature *leaf dropping (leaf abscission, leaf casting, defoliation)*. It cannot be described in morphological terms but finds its origin in correlative disturbances. This phenomenon is rather common in virus diseases, but is very striking in sour cherry with cherry yellows (KEITT & CLAYTON, 1943). It typically begins

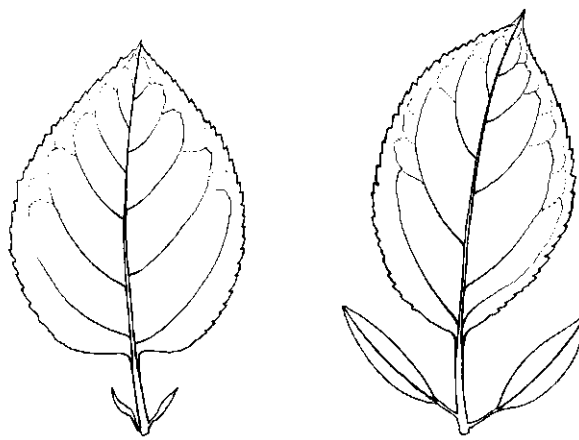


Fig. 25. Hypertrophy of stipules in apple leaves caused by the apple witches' broom virus. Left, normal leaf. (Drawing Miss I. Zewald, Department of Plant Taxonomy and Plant Geography, Agric. Univ., Wageningen).

with the older leaves and extends towards the younger. Leaves may be cast before any chlorosis is visible and defoliation may amount to 50% of the total number of leaves.

Leaf dropping in some potato varieties due to potato virus Y (sometimes called 'potato leaf drop streak') is of a different origin. This type of leaf dropping is associated with wilting and withering of the leaf. The hanging position of the leaf is primarily due to necrosis extending down the petiole.

For a number of deformities due to secondary local growth reductions in the leaf cf. p. 84 and 85.

Enations were mentioned earlier as histoid deviations, but a number of enations on leaves should be classified as organoid deviations, because they have the internal structure of a normal leaf. These organoid *enations* usually develop on the under surface of the leaf. They are often associated with chlorotic areas, which they surround as fringes, and sometimes consist merely of rough ridges. Quite often, however, the enations are leaf-like structures composed of an upper epidermis, a palisade layer, a spongy parenchyma and a lower epidermis. The origin of enations around chlorotic areas suggests a loss by these areas of their normal morpho-genetic control over surrounding tissues (KUNKEL, 1954). As a consequence, the green edges grow out as leaf edges, developing into cup-like structures around the chlorotic fleck and having an upper surface and palisade layer on the inside. This type of enation occurs in *Nicotiana paniculata* and *N. tomentosa* after infection with the ordinary tobacco mosaic virus (JENSEN, 1933). It is also very common in pea, broad bean, and crimson clover after infection with the pea 'enation mosaic' virus (e.g. MCWHORTER, 1950). The morphology, anatomy and histogenesis of the enations on the leaves of *Nicotiana glutinosa* and *Lycopersicum esculentum*, infected with the aspermy virus (a strain of the cucumber mosaic virus) (fig. 26), have been studied recently by PRACEUS (1958). These enations occur interveinally or in the neighbourhood of the veins but very seldom on the veins. They may range in form from leaf-, wing-, cup-, boat-, or funnel- to shell-like structures. Often they protrude a few millimeters from the lower surface of the leaf. Their position on the leaf is apparently at random. According to NOORDAM (1952) the aspermy virus produces enations even in the corolla of *Petunia hybrida*. The funnel- or cup-shaped

Fig. 26. Organoid enations in Nicotiana rustica caused by the aspermy virus. (After KRISTENSEN & THOMSON, *1958).*

enations caused by the tobacco leaf-curl virus (kroepoek virus) in tobacco leaves are initiated on the under surface of the veins (KERLING, 1933). The same holds for the enations on cherry leaves due to the Pfeffinger- or Eckelrader-disease virus (STOLL, 1952). They originate on small side veins in the neighbourhood of the main vein.

Deviations in the morphology of the stems also may be due to local growth reductions. Virus-diseased plants may show a *shortening of internodes*. Branches of grape-vine affected by fan-leaf disease usually show short internodes; some of them may be entirely lacking, leading to the so-called *double nodes* (HEWITT & GIFFORD, 1956). When this reduction concerns all internodes of a stem, the leaves become more or less rosetted. Infections at a late stage of plant development may result in a conspicuous crowding of the leaves at the extremities of the branches. Some examples of virus diseases characterized by this *rosetting* of leaves are apple 'rosette', peach 'rosette', Pfeffinger or Eckelrader disease of cherry (in German sometimes called 'Rosettenbüschelkrankheit'), and groundnut 'rosette'. The bushy appearance may sometimes produce some confusion with witches' broom formation. However, this is an entirely different symptom (cf. p. 76). Usually the leaves involved also show some abnormalities in rosette diseases.

Another morphological stem disorder is the peculiar *zigzag growth* of grape canes in vines with fan leaf. Together with other malformations, such as short internodes and double nodes, this

phenomenon may be of diagnostic value when the vines are dormant and the leaves cannot be observed (HEWITT & GIFFORD, 1956).

The *aerial tuber formation* in potato plants is not a common phenomenon (fig. 27). It may occur as a result of infection by aster yellows virus (SEVERIN, 1940), by stolbur virus (cf. a survey of literature by KLINKOWSKI, 1958) or by potato witches' broom virus (TODD, 1958).

A very peculiar effect of a virus on the morphology of fruits is caused by the quercina virus (tobacco severe etch) on the fruits of thorn-apple *(Datura stramonium)*. The spines on the capsules

Fig. 27. Aerial tuber formation in potato plant infected with an unidentified witches' broom virus related to stolbur virus and to aster yellows virus.

are partially or entirely suppressed because of the virus infection (BLAKESLEE, 1921). Recently it has been reported that cucumber mosaic virus has the same effect in this host (AUBERT, 1960).

The *witches' broom phenomena* comprise an extremely interesting group of organoid modifications due to viruses. The symptoms are caused by a complex of changes in vegetative and sexual parts of the plant and they influence the morphology of the whole plant. The phenomena consist of witches' broom growth and a complex of floral abnormalities. Their patho-morphology has been studied thoroughly by Bos (1957a), who gives a survey of a large number of witches' broom viruses inducing these phenomena. Among them are listed virus diseases such as 'aster yellows', 'clover virescence', 'tomato big bud', 'stolbur' and 'Rubus stunt'.

Fig. 28. Witches' broom growth in Vaccinium myrtillus; left, healthy plant; right, naturally infected plant. In contrast to the healthy plant the diseased plant kept its leaves in the greenhouse during winter. (After Bos *1960).*

The symptoms depend to a high degree on the stage of development of the plant at the moment of infection and to some extent on the host plant species. Thus, after an early infection, vegetative abnormalities such as witches' broom growth may predominate, whereas floral abnormalities may prevail after a late infection during or at the beginning of flower initiation. This often led to confusion in the naming of these diseases. Inaccurate or confusing names and synonyms will be mentioned in the following text. In view of this confusion and the lack of information in the literature, the patho-morphology of these witches, broom phenomena will be described in some detail.

Because of an excessive and premature development of buds, oftentimes of adventitious buds, leading to extravagant branching, the plant develops a bushy, broomy appearance. Since this systemic brush-like growth is comparable to the formation of local brooms, such as those caused by mites or fungi in trees and other plants and known as witches' brooms, the abnormal type of growth incited by viruses is called *witches' broom growth* (fig. 28). The erect position of the newly formed branches, indicative of an *intensified negative geotropy*, contributes to the broom-like habit. For this phenomenon the names *blastomania* and *cladomania*, adopted from teratology and referring to the mania or rage to develop buds and branches, are correct (Gr. blastos = sprout, but also sprouting or shooting; klados = branch).

The term blastomania has been used only once in virus literature, viz. by THIEM (1956) for the witches' broom disease of apple. Because of the production of an abnormal number of branches, this witches' broom disease of apple, has generally been called 'apple proliferation disease' since MULDER (1953) introduced this name. This name is confusing for several reasons, and it is suggested here that it be omitted. The term *proliferation* (cf. p. 59) is generally used in biology for an uncontrolled, almost unlimited and abnormal development and multiplication of cells, tissues and organs. Although it could be said that a proliferation of branches occurs in this disease, the name 'proliferation disease' does not specifically indicate whether cells, tissues or specific organs proliferate. Proliferation literally means the 'bearing' of a sprout or of sprouts. The development of axillary buds into a vegetative sprout in itself is not abnormal. However, sprouting in excessive numbers is abnormal. For this phenomenon the terms *blastomania* and *cladomania* are correct and self-explanatory. (For a discussion of the distinction between proliferation and prolification cf. p. 81).

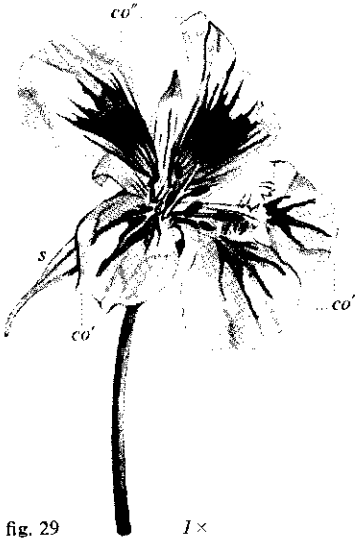


fig. 29 *1* ×

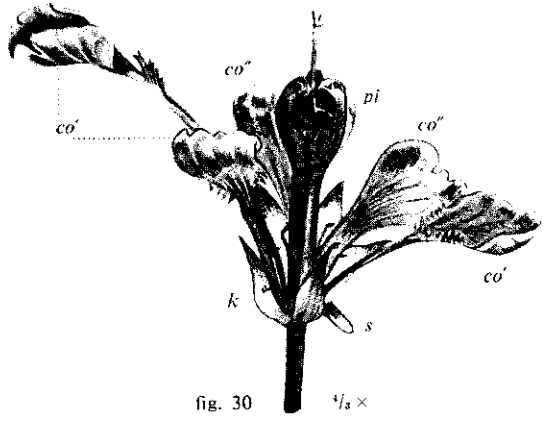
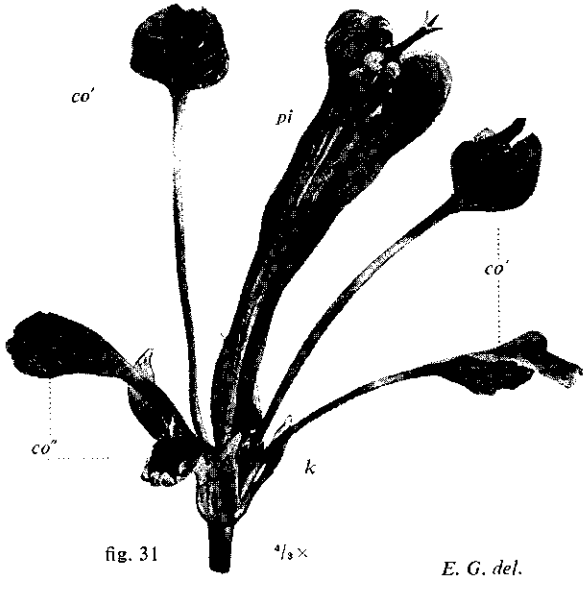


fig. 30 $^{4}/_{5}$ ×



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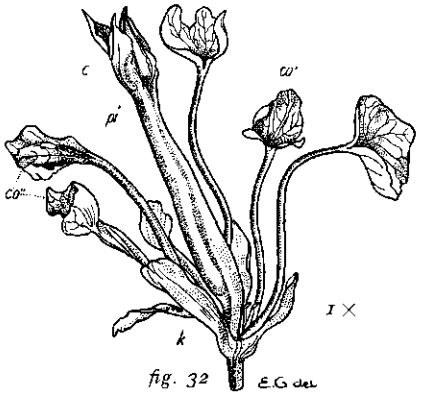


fig. 32

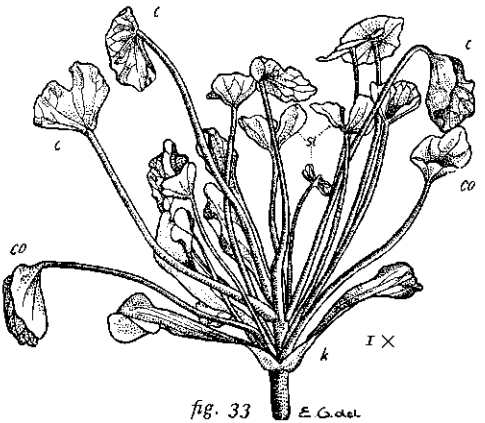


fig. 33

½ ×

fig. 34

Legend see page 79

on the stage of flower initiation at the time of infection or more precisely at the time when sexual processes of the plant are disturbed. The floral abnormalities led to the name 'false blossom' (SHEAR, 1916) for a witches' broom disease of the cranberry.

The process of antholysis starts with *virescence* or *greening* (L. virescere = to grow green) of normally white or coloured floral parts through the development of chloroplasts (cf. also colour deviations of flowers) (fig. 29–31). This phenomenon is characteristic of such diseases as 'virescence of tomato' (a syno-

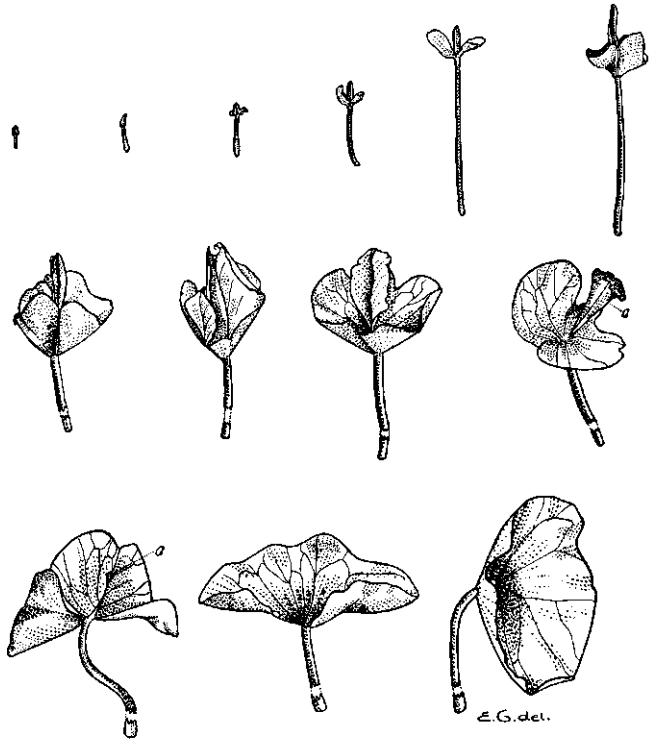


Fig. 35. Phyllody of stamens in Tropaeolum majus caused by a witches' broom virus. Top left, normal stamen; bottom right, final result of phyllody, normal peltate leaf. Magn. × *1. (After* BOS, *1957a).*

nym of 'tomato big bud' (HILL, 1943)), 'green flowering disease' of *Justicia gendarussa* in Birma (SU, 1933) and of *Sesamum indicum* in India (ROBERTSON, 1928), and 'green petal' of strawberry (POSNETTE, 1953).

When the floral parts develop as more or less normal foliage leaves in later stages of antholysis, the phenomenon is called *phyllody* (Gr. phyllon = leaf) (fig. 30–34). In sepals this condition is often associated with considerable *hypertrophy*, such as in flowers of raspberry infected with the Rubus witches' broom (Rubus stunt) virus. In tomatoes infected with stolbur or tomato big-bud virus the calyx is very much enlarged, even before opening of the flower bud. This leads to the 'big (flower) bud' symptom. Stamens are rather seldom phylloid, but they also may show interesting phyllody such as in *Tropaeolum* (fig. 35) (BOS, 1957a). Phyllody of carpels is especially very striking. The enlarged open carpels dominate the floral picture and protrude beyond the virescent inflorescence such as in clover with 'clover phyllody' or 'clover virescence'. The ovules also are often present as small leaf-like organs at the edge of the open carpel (fig. 36). On this basis witches' broom virus diseases sometimes were named 'phyllody', such as 'phyllody of sann hemp' *(Crotalaria juncea)* in India (BOSE & MISRA, 1938) and 'phyllody' of *Sesamum indicum* in India (PAL & NATH, 1935).

The next step in antholysis is *apostasis*, which means the development of internodes theoretically present in the receptacle, which is the part of the flower in which the floral parts are inserted (Gr. apo = away from, without; Gr. stasis = a standing still or stoppage, arrested motion or action). Usually this only concerns the stipe or gynophore, which is the internode between the insertion of stamens and carpels (fig. 34 and 36). In this way

Fig. 29 - 34. See colour plate and page 77. *Antholysis in Tropaeolum majus, caused by a witches' broom virus and consisting of virescence (fig. 30 and 31), phyllody (fig. 30 - 34), and prolification (fig. 34), (fig. 29 normal flower). Note especially the phyllody of stamens in fig. 33 and of the carpels particularly in fig. 32 - 34, and the prolification in fig. 34 on top of the torus and in the axils of the phylloid carpels. k = calyx, s = spur, co = petals (co' = lower three, co" = upper two), st = stamens, pi = pistil, c = carpel, g = stipe. (After* BOS, *1957 a).*

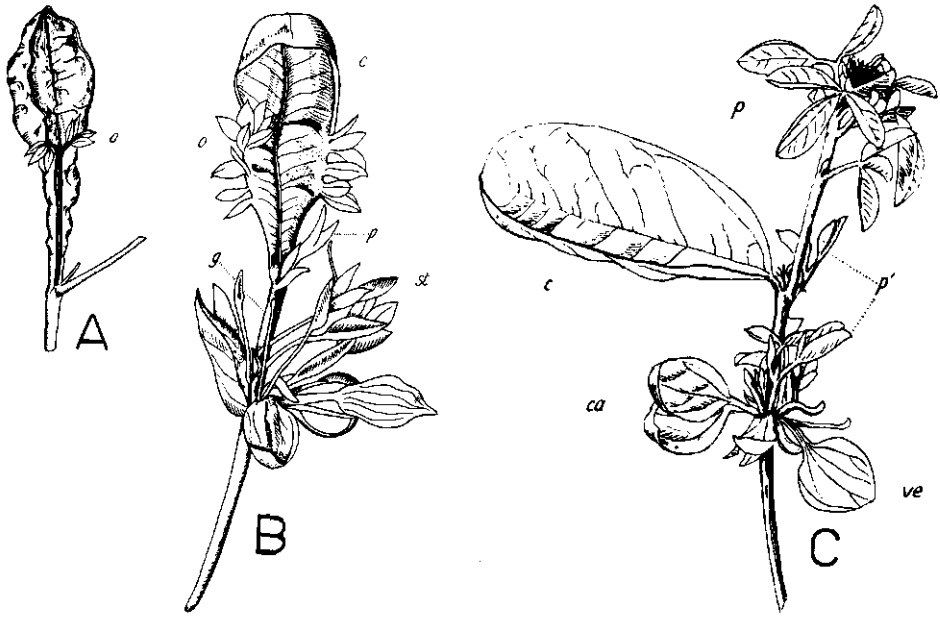


Fig. 36. Phyllody of carpel (c) *and ovules* (o) *in Crotalaria usaramuensis caused by a witches' broom virus. In B and C also central* (p) *and lateral* (p') *prolification occurs.* g *gynophore or stipe. (Magnification A, B,* $\times 2$, *C* $\times {}^{4}/_{3}$*). (After* Bos, *1957a).*

the abnormal pistil is raised beyond the flower. This contributes to the protrusion of the phylloid carpels beyond the inflorescence, as mentioned above.

The elongation of the receptacle above the insertion of the pistil(s) and development into a sprout is quite common. In teratology this phenomenon has long been known under the name of *prolification*, which literally means the production of a sprout or sprouts (L. proles = offspring, sprout; L. ficare = to make) (fig. 34, 36B, C). In addition to this *central prolification* from the apex of the receptacle, *lateral prolification* may also occur. This is the development of sprouts from the axils of the floral organs (fig. 36C). For this phenomenon in plant teratology originally both the terms proliferation and prolification have been

used. Both have a similar literal meaning with the only difference that proliferation (cf. also p. 59) means the 'bearing' of a sprout or of sprouts, and prolification means the 'making' of a sprout or of sprouts. Since in the literature proliferation has been applied for uncontrolled sprouting of cells and also for rather unlimited development of tissues and multiplication of organs, the term prolification is preferred for the 'sprouting' of flowers. The newly formed sprouts usually are vegetative, often much ramified leafy branches. A new inflorescence or flowers may also develop especially in the case of central prolification. Usually these new flowers are atypical. In this way a series of abnormal flowers may occur, with one developing above the other. It is evident how this prolification of flowers makes the inflorescence into a witches' broom and contributes greatly to the witches' broom growth of the plant. The vegetative type of floral prolification is fundamentally similar to the witches' broom growth in the vegetative part of the plant, both being due to the excessive and premature development of all actually and potentially present buds.

Finally we may find entirely vegetative sprouts instead of flowers, e.g. in the axil of a bract in the upper part of an abnormal inflorescence. This represents the last stage of the whole series of antholysis. Plants infected early during their development often fail entirely to produce flowers. It is clear that the witches' broom phenomena lead to complete *sterility* of the infected plants (cf. the name 'sterility' of *Sesamum indicum* in India (ROY, 1931)). This characteristic makes the witches' broom virus diseases of particularly great economic importance. Infected plants do not produce seeds or fruits. For this reason such names as 'diseases causing seedlessness in plants' (BOJNANSKY & BLATTNY, 1953) or 'diseases causing sterility' (cf. Proceedings of the scientific conference on stolbur and similar diseases causing sterility, 1958) have been invented for some witches' broom virus diseases. Even the German name 'Akarpien' ('diseases characterized by fruitlessness') has been proposed (BLATTNY, 1959).

All witches' broom virus diseases studied thoroughly so far showed clearly an association of witches' broom growth and antholysis. As already described above, the floral abnormalities even contribute to the witches' broom growth. The dominance of certain symptoms depends greatly on the stage of develop-

ment of the plant at the time of infection [1]. The witches' broom growth attracts most attention after infection during vegetative development. Infection during flower initiation, however, leads to a prevalence of floral abnormalities. This has often given rise to names, mentioned above, suggesting different diseases. Since all of these symptoms always belong to the same complex of *witches' broom phenomena*, it has been suggested (Bos, 1957a) that the name 'witches' broom virus diseases' be applied to this group of virus diseases.

A number of special deviations due to these viruses still need to be mentioned here. In *Phaseolus calcaratus* a germination of seeds in immature pods already developed before infection has been observed (Bos, 1957a). This suggests a *reduction of the seed dormancy*. It indicates a *disturbed periodicity* in these affected plants. This disturbance is also obvious from the fact that

[1] Frazier & Posnette (1957) reported to be able to distinguish between a virus causing only phyllody in clover and a virus causing only witches' broom growth in the same host. This has not yet been supported by others.

Fig. 37. Witches' broom growth and bolting in young carrot plants after artificial infection with a witches' broom virus of clover. Left, healthy plant.

witches' broom virus diseased plants tend to keep growing in autumn and to start their development earlier in spring than normal plants do. This is characteristic of the witches' broom disease of *Vaccinium myrtillus* (Bos, 1960). Another striking example of the disturbed periodicity is that of carrot plants flowering the first year (although abnormal flowers are produced) instead of during the second year as in normal plants, after infection with a witches' broom virus of clover (Bos, unpublished) (fig. 37).

The *intensification of negative geotropy* due to virus, which leads to an erect position of newly formed branches as mentioned before, makes the normally positive geotropic stipes of peanut plants negative geotropic. Thus, in plants infected while flowering and forming pods, the stipes may curl upwards after having curled downward, even after they have penetrated the soil and started pod formation. The same phenomenon can be observed in the pedicels of tomato plants infected with stolbur or tomato big-bud virus. Instead of bending downwards, the pedicels show an upright position. The same holds for the pedicels of cranberry infected with false-blossom virus (SHEAR, 1916).

Potato plants may produce an exceptionally large number of small tubers, e.g. after infection with the potato witches' broom virus (TODD, 1958). In this plant species even *aerial tubers* may be formed after infection with the same and some related viruses (cf. p. 73 and fig. 27). Tubers of potato plants infected with stolbur virus (SAVULESCU & POP, 1956), potato witches' broom virus (TODD, 1958), aster yellows virus (LARSON, 1959), or tomato big-bud virus (WEBB & SCHULZ, 1958), often show *spindle-sprouting* or *hair sprouting* after germination and produce spindling, spindly or hair sprouts. According to LARSON (1959) this only occurs under conditions of high soil temperature after planting. With a low temperature, on the contrary, only short sprouts are formed, producing small secondary tubers at their extremities in the neighbourhood of the old tuber.

For a discussion of the physiological and hormonal disturbances underlying the induction of witches' broom phenomena cf. Bos (1957a).

B. The *secondary malformations* are due to other abnormalities which find their origin primarily in the virus infection. These

secondary phenomena are quite common in virus diseases. As will be described below, they may strongly influence the external shapes of the plant, without qualifying for classification among histoid or organoid deviations.

Here also developmental proportions may be disturbed because of differences in growth rate. Discoloured parts of a leaf often have reduced vitality. In leaves with an irregular mosaic pattern this may lead to internal tensions. If the mosaic spots occur near the edges of the leaf, these local tensions may lead to deeper incisions of the margin or to an irregular lobation. Thus diseased leaves often have a very irregular shape which cannot be described in morphological terms. In many cases, however, the tensions only find an outlet in a direction perpendicular to the flat surface of the leaf and result in symptoms such as those induced by the tobacco mosaic virus in tobacco leaves (fig. 38) and by the common bean mosaic virus in the leaves of French beans. In the latter case the dark-green areas of the leaf, mostly situated along both sides of the main vein, have a faster rate of growth than the surrounding tissue and bulge caterpiller-like.

A similar phenomenon of deformation starting as a mosaic may be observed in fruits, e.g. in gherkins after infection with cucumber mosaic virus (fig. 39) (TJALLINGII, 1952).

Fig. 38. Secondary leaf deformation of White Burley tobacco as a consequence of mosaic formation caused by tobacco mosaic virus.

Fig. 39. Secondary deformation in fruits of gherkin infected with cucumber mosaic virus. (After TJALLINGII, *1952).*

Several other leaf deformations, such as *rugosity, crinkling* and *curling* were already ascribed to a reduced growth rate of the veinal tissue (p. 69). This growth reduction of the veins may also be due to other virus symptoms such as veinal necrosis. This secondary curling and crinkling generally is of a more irregular type than that occurring as a result of a growth reduction regularly distributed over the veinal system.

For the same reasons as mentioned above the internal forces in the leaf may be torsive leading to *distortion.*

Obviously many malformations of leaves also may be due to localized necroses. Since the necrotic tissue ceases to grow, tensions are induced in the surrounding tissues which are still developing. This phenomenon is very pronounced in tobacco systemically infected with rattle virus.

Often a necrosis of the stem apices occurs, with the result that many lateral branches may develop, giving the plant a bushy, witches' broom-like appearance. In turn many axillary shoots may develop abortive apices, and then further branching takes place e.g. in tomato aspermy (BLENCOWE & CALDWELL, 1949).

Localized necrosis and excessive formation of sclerenchyma in stony pit of pear also leads to malformation of the pear fruit.

VIII. Virus particles and inclusion bodies

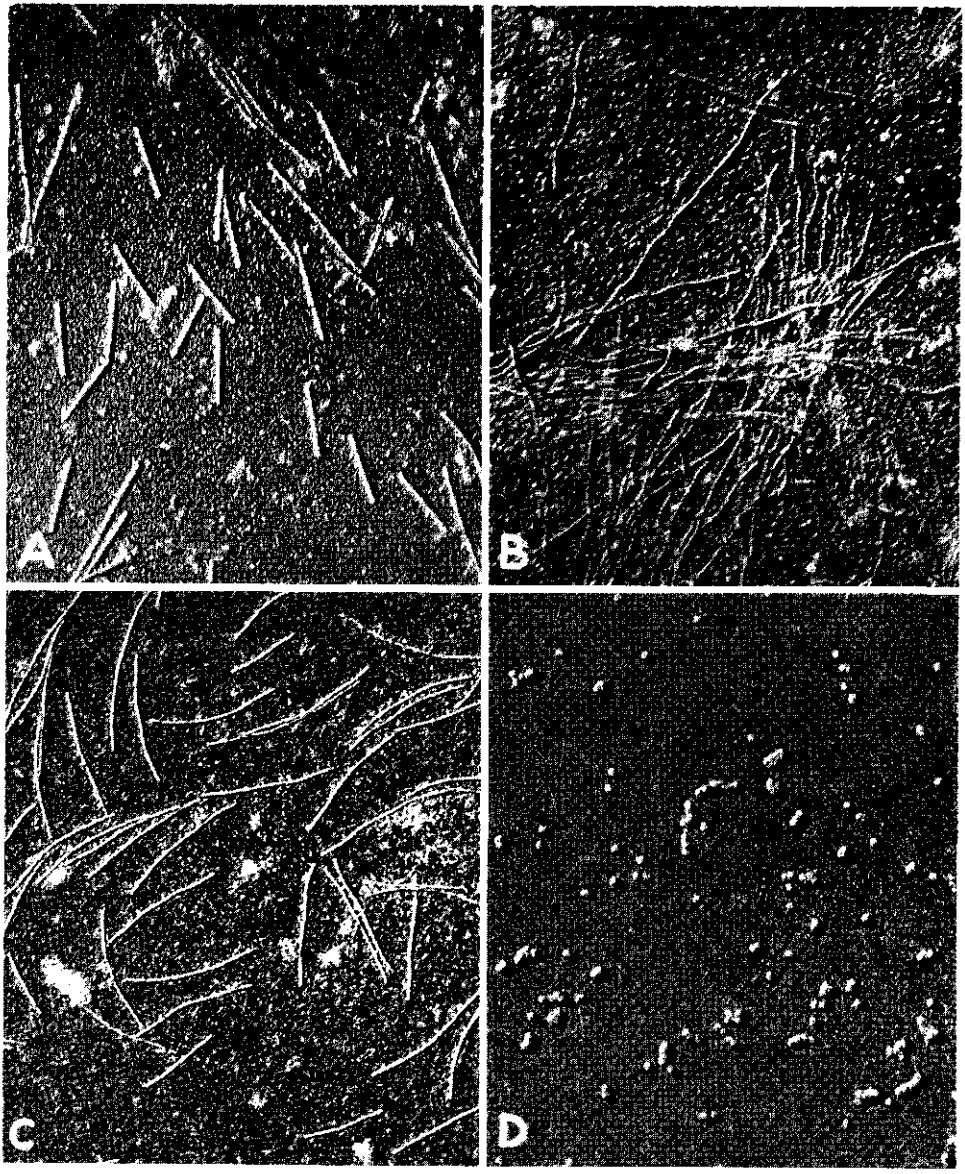
For a very long time, the incitant of virus diseases escaped observation. In the Pasteur and Koch period the general assumption was that infectious diseases had to be due to visible organisms. This assumption probably delayed the detection of the nature of virus diseases for a long time. Without demonstration of a visible incitant, nevertheless, the infectious nature of many virus diseases was established during the first decades of plant virology.

The absence of a visible causative agent made virus diseases entirely different from the other contagious diseases. Generally the absence of a visible incitant in the case of an infectious disease was the most important argument leading to the conclusion that a virus disease was involved. So in contrast with fungi and bacteria, viruses themselves were assumed to take no part in the visible symptoms. Fungi may form a characteristic mould or fruiting bodies on or in infected parts of the host. Similarly bacteria may form typical colonies. In this way these diseases often may be easily recognized.

In 1939 KAUSCHE, PFANKUCH & RUSKA, however, for the first time observed virus particles by means of an electron microscope, thus demonstrating directly their corpuscular nature. Since then, the number of viruses which have been studied for their morphology and size is ever increasing. Thus far, this type of study has been restricted to those viruses which have been transmitted mechanically to plant or to insect vector.

The form and size of plant viruses appear to be constant within certain limits and therefore are very characteristic. The particles may be spherical or polyhedral, rod-shaped, and thread-like. They may vary in size from 16 to about 110 mμ for the so-

Fig. 40. Electron micrographs of four plant viruses. A. tobacco mosaic virus, B. pea mosaic virus, C. red clover vein-mosaic virus, D. red clover mottle virus. Magn. x 27,000 (Photographs made by the Physico-Technical Service for Agriculture, Wageningen). ▶


A
B
C
D

called spherical viruses and from 55 to 1250 mμ for the elongated. The width of these elongated particles varies from about 10 to 30 mμ. Electron micrographs of some particle types are given in fig. 40.

Opinions may differ as to whether these facts should be mentioned in a publication on symptoms of virus diseases. Since the virus particles themselves also are products of the interaction between plant and virus, they have to be taken into consideration here. Moreover, their presence contributes to the abnormality of the host, and with modern techniques they sometimes can be observed rather easily. In this connection the very rapid method of making preparations for the electron microscope developed by BRANDES (1957) is noteworthy. The cut surface of a diseased plant part is dipped into a small drop of water on the specimen holder for one to two seconds only. After drying, the preparation is ready for shadow casting and then for being studied in the electron microscope. The preparation of fig. 40 C has been made according to this method. Since new and improved techniques in the future undoubtedly will be developed, the observation of virus particles in diseased plant parts will be of increasing importance. The morphology and size of these particles belong to the intrinsic characters of the incitants. Their study is therefore of special importance from a diagnostic point of view. For a further discussion of this aspect cf. BRANDES & PAUL (1957) and BRANDES & WETTER (1959).

In this connection the so-called *inclusion bodies* are especially important. They represent a peculiar and characteristic phenomenon involved in a number of virus diseases. After proper staining they are visible with the light microscope, especially in epidermal and hair cells, and have a size ranging from about 5 to 30 μ or even smaller. In some diseases, the morphology and location of these bodies may contribute to the identification of the virus. The inclusions can be stained in several ways, e.g. with trypan blue.

Inclusion bodies have been known for a very long time. IWANOWSKI (1892, 1903) discovered two types of inclusions in the cytoplasm of mosaic-diseased tobacco, viz. *amorphous inclusions*, the so-called X-bodies, and *crystalline inclusions* (fig. 41). Both types of inclusions may occur independently, but changing of the first into the latter type has also been observed (KASSANIS &

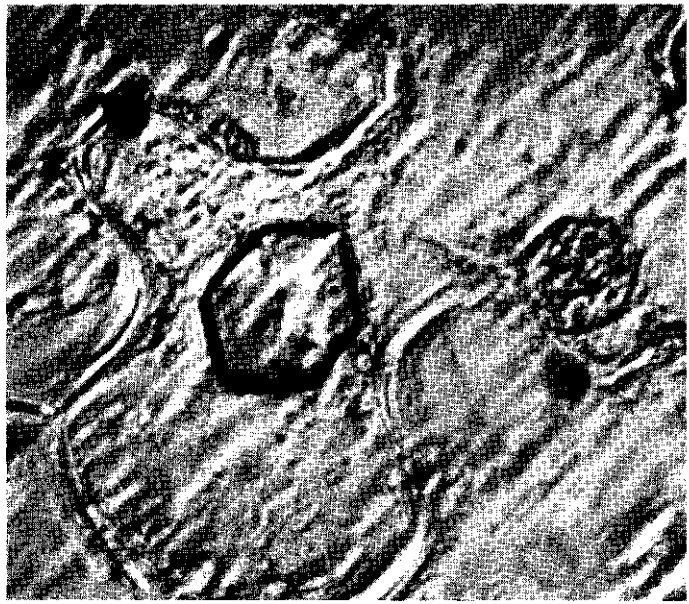


Fig. 41. Crystalline inclusion body in epidermal cell of Vicia faba infected with the red clover vein-mosaic virus. (Photo courtesy Dr. C. A. Porter, Yonkers, N.Y.).

SHEFFIELD, 1941). Their discovery in cactaceous plants by MOLISCH (1885) eventually led to the detection of the associated and first-known virus disease of these plants, the incitant being infectious and filterable (ROSENZOPF, 1951) and being rod-shaped and a nucleoprotein (AMELUNXEN, 1958). Here the changing of granular X-bodies into crystalline inclusions has also been observed and described in detail (AMELUNXEN, 1958). The presence of crystalline bodies within the nucleus, *intranuclear inclusions*, is rather rare in plant virus diseases. They do occur, however, as in tobacco and some other plant species with severe etch disease (KASSANIS, 1939).

The amorphous inclusions are generally irregularly globular or amoeboid in shape. They can be easily mistaken for the nucleus. The crystalline intracellular inclusions vary in shape

from irregular hexagonal plates (tobacco mosaic virus) and isometric crystals (pea mosaic virus and bean yellow mosaic virus in broad bean) to spindle-shaped bodies (cactus virus) or even needle-shaped fibres (tobacco mosaic virus).

The crystalline intranuclear inclusions also vary in size and shape. There may be more than one in the same nucleus, such as in those of leguminous plants infected with pea mosaic and bean yellow mosaic virus, where more than five may be present (McWHORTER, 1941).

For some time the inclusions have been assumed to consist of virus protein (SHEFFIELD, 1946), whereas in X-bodies other constituents also may occur (SHEFFIELD, 1931). By intact removal of the crystalline inclusion bodies from hair cells of tobacco plants infected with tobacco mosaic virus by a freeze-drying technique and dissection of the crystals, STEERE & WILLIAMS (1953) could demonstrate with the electron microscope that the crystals consist apparently of nothing but virus particles and a volatile solvent. By means of the electron microscopy of ultrathin sections, BRANDES (1956) was able to demonstrate that the X-bodies in mosaic-diseased tobacco plants are largely composed of virus particles in an irregular arrangement apparently surrounded by a membrane. In contrast, the crystalline inclusions show a regular crystalline arrangement of virus particles. The protein spindles of cactaceous plants were also shown to be composed of infectious virus particles in a distinct arrangement (AMELUNXEN, 1958). How the virus particles aggregate to these structures is not yet known. The name 'viroplasts', used by McWHORTER (1941) for the inclusion bodies, clearly indicates their virus nature.

This makes clear that virus particles themselves may produce microscopically visible deviations and that the virus particles themselves may contribute to the complex of disease phenomena in virus-infected plants.

A rather extensive literature on virus inclusions has been published. BAWDEN (1950) lists 22 viruses, which are able to produce these bodies. A recent short survey of literature is given by SMITH (1958).

IX. Phenomena due to secondary causes

A general reduction in vitality often occurs in virus-diseased plants. This may lead to a visible reduction in plant size (cf. section on growth reduction). In other cases the result may be invisible to the eye and consist only of physiological weakening of the plant. This weakening often becomes visible only under unfavourable conditions. Thus, Ladino clover plants infected with lucerne mosaic virus appeared to suffer more from frost than did healthy ones (ROBERTS, 1956).

Because of this weakening, virus-diseased plants also may have an increased susceptibility to secondary infections with fungi and bacteria. Peas diseased with leaf roll (tip yellows), for example, often are infected with the soil-inhabiting *Fusarium solani*. Until recently, this fungus was even assumed to be the incitant of the disease. However, it is now known that this fungus is unable to attack a healthy pea plant (HUBBELING, 1954). Broad bean plants infected with the same leaf-roll virus show an increased susceptibility to *Botrytis fabae* in comparison with healthy ones (TINSLEY, 1959). According to HEILING *et al.* (1956), infection of sugar beets with yellows virus promotes infection with the fungus *Cercospora beticola*.

Rotting sometimes accompanies virus diseases. This, however, is never incited by the virus itself, but is due to a secondary infection by fungi or bacteria, which are able to invade weakened, dying or necrotic tissue. Bacteria cause a decay of the dead tissue called wet rot. In contrast, fungi usually produce a dry rot such as in stolbur-affected potato plants which show a marked predisposition to infection with *Colletotrichum atramentarium* (KOVACHEWSKY, 1954; WENZL, 1956). The above-mentioned secondary infection of leaf-roll infected peas with *Fusarium solani* leads to foot rot.

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Index of names of symptoms in five languages

The terms considered the most appropriate are always mentioned first. It is suggested that the names in parentheses be omitted in virological terminology. However, they are given here because they can be found in older publications or are still being used occasionally. Their listing here also makes the key to the text a complete one. Names in quotation marks are used more to indicate diseases than symptoms.

ABBREVIATIONS:
d = Dutch; **g** = German; **f** = French; **i** = Italian.

I. English index with Dutch, German, French, and Italian equivalents

antholysis 76
d antholyse, bloemoplossing
g Antholyse
f antholyse
i antolisi

apostasis 79
d apostasie
g Apostasie
f apostasis
i apostasi

asteroid spotting 42
d stervormige vlekking
g Sternfleckung
f taches étoilées
i macchiettatura asteroide

atrophy 27
d atrofie
g Atrophie
f atrophie
i atrofia

(aucuba mosaic) 38
d (aucubamozaïek, aucubabont)
g (Aucubamosaik)
f (mosaique aucuba)
i (mosaico aucuba)

---

bark necrosis cf. *canker*

bark scaling 57
d schubbigheid
g Schuppigkeit
f desquamation corticale
i desquamazione della corteccia

blackening 32 45
d zwartkleuring
g Schwarzfärbung
f noircissement
i annerimento

blanching, bleaching 32
d verbleking, witkleuring
g Bleichung ('Bleiche')
f blanchiment
i sbiancatura

blastomania cf. *witches' broom growth*

bleaching cf. *blanching*

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d bloemkleurbreking
g Blütenfarbenbrechung, (Buntstreifigkeit)
f panachure de la fleur
i rottura del colore dei fiori

bronzing 32 45 52
d bronskleuring
g Bronzefärbung
f bronzage
i bronzatura

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g Braunfärbung, ('Bräune')
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d (calico)
g (Kaliko)
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i (calico)

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d (overmatige) callosevorming
g (übermässige) Kallosebildung
f formation (excessive) de callose
i formazione (eccessiva) di callosi

canker, bark necrosis 54
d bastnecrose, baststerfte
g Rindenbrand
f nécrose corticale
i necrosi corticale

cankerous tumour 65
d kanker (partly)
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d chlorose
g Chlorose
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d chronische fase
g chronische Phase
f phase chronique
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cladomania cf. *witches' broom growth*

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d kurkvorming
g Korkbildung
f subérisation
i suberosi

crinkling, crumpling, wrinkling 69 85
d kreuk(el)ing, rimpeling, (krinkeling)
g Kräuselung, leichte Kräuselung
f frisolée faible, gaufrage
i arricciamento leggero

curling, leaf curling 69 85
d krulling, bladkrulling
g Kräuselung, schwere Kräuselung
f frisolée grave
i arricciamento grave

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d aftakeling
g Abzehrung, Verfall
f dépérissement
i deperimento

defective lignification 29
d onvolledige verhouting
g unvollständige Verholzung
f lignification défectueuse
i lignificazione incompleta

defoliation cf. *leaf dropping*

deformation cf. *malformation*

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d verdroging, (uitdroging)
g Vertrocknung (Austrocknung)
f dessèchement
i disseccamento

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d ziekte
g Krankheit
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d verwringing, distorsie
g Verdrehung, Verkrümmung
f distorsion
i distorsione

disturbed periodicity 82
d verstoorde periodiciteit
g gestörte Periodizität
f altération de la périodicité
i alterazione del periodismo

dotting cf. *speck(l)ing*

double nodes 72
d dubbele knopen
g Doppelknoten, (Kurzknoten)
f doubles noeuds
i nodi doppi

dwarfing, stunting 29
d dwerggroei
g Zwergwuchs, Kümmerwuchs, Nanismus, Stauchung, Verzwergung
f nanisme
i nanismo

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d randchlorose, bladrandchlorose
g Randchlorose, Blattrandchlorose
f chlorose du bord de la feuille
i clorosi marginale

edge yellowing 33
d randvergeling, bladrandvergeling
g Randvergilbung, Blattrandvergilbung
f jaunissement du bord de la feuille
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d enatie, uitwas
g Enation, Auswuchs
f énation
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d etsing
g Ätzung, Ätzmuster
f gravure
i 'etching'

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g Fächerblattbildung
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d takafplatting
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d vergroening, virescentie
g Vergrünung, Vireszens
f virescence
i inverdimento, virescenza

growth reduction 29
d groeiremming
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g Gummosis, Gummibildung
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d hypoplasie
g Hypoplasie
f hypoplasie
i ipoplasie

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g Immunität
f immunité
i immunità

inapparency, inapparent infection 14 16
d inapparentie, symptoomloosheid, inapparente infectie, symptoomloze infectie
g Inapparenz, inapparente Infektion
f infektion inapparente
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inapparent infection c.f *inapparency*

inclusion bodies, (viroplasts) 29 88
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g Einschlusskörper
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i periode d'incubazione

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d infectie
g Infektion
f infection
i infezione

inoculation 13
d inoculatie, inenting
g Impfung (Beimpfung eines Blattes, Verimpfung eines infektiösen Saftes)
f inoculation
i inoculazione

internode shortening, shortening of internodes 72
d internodiënverkorting, stengellidverkorting
g Internodienverkürzung
f 'court-noué'
i accorciamento degli internodi

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d tussennervig mozaïek
g zwischennerviges Mosaik
f mosaïque internervale
i mosaico internervale

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d latentie
g Latenz
f latence, état latent
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leaf abcission cf. *leaf dropping*

leaf casting cf. *leaf dropping*

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g Blattfall
f abscission des feuilles
i filloptosi, caduta di foglie

leaf narrowing 67
d smalbladigheid
g Schmalblättrigkeit
f rétrécissement du limbe
i restringimento della foglia, stenofillia

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d bladrolling
g Blattrollung
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g Linien- und Kurvenmuster
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f lésion locale
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g Lokalsymptomen
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i malformazione, deformazione

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d maskering
g Maskierung
f masquage
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g Mosaik
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g diffuse Fleckung?
f marbrure
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g Nekrose
f nécrose
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g Verstärkung der negativen Geotropie
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g (Teratomata)
f (tératomes)
i (forme teratologiche)

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d tolerantie
g Toleranz
f tolérance
i tolleranza

top necrosis 53
d topnecrose, topsterfte, (acronecrose)
g Spitzennekrose, (Akronekrose)
f nécrose apicale
i necrosi apicale

tumorous canker 55 65
d kanker (partly)
g Krebs (partly)
f chancre (partly)
i cancro (partly)

tumour (Am. tumor) 62
d tumor, gezwel
g Tumor
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tylose formation, (excessive) – 27
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g (übermässige) Thyllenbildung
f formation (excessive) de thylles
i tillosi, eccessiva formazione di tilli

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d bontheid
g Buntblättrigkeit
f variégation
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g Adernnekrose, ('Rippenbräune')
f nécrose des nervures
i necrosi delle nervature

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g Adernbänderung, Adernbandmosaik
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vein chlorosis 33 37
d nerfchlorose
g Adernchlorose
f chlorose des nervures
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d nerfglazigheid
g Adernaufhellung
f éclaircissement des nervures
i schiarimento delle nervature

vein mosaic 39
d nerfmozaïek
g Adernmosaik
f mosaïque des nervures
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vein yellowing 33
d nerfvergeling, (geelnervigheid)
g Adernvergilbung
f jaunissement des nervures
i ingiallimento delle nervature

virescence cf. *greening*

(viroplasts) cf. *inclusion bodies*

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water deficiency, water shortage 27 49
d watertekort, watergebrek
g Wasserdefizit
f déficience en eau, manque d'eau
i insufficienza idrica, deficienza idrica

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d verwelking
g Verwelken, Welken, ('Welke')
f flétrissement
i appassimento

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d heksenbezemvorming
g Hexenbesenbildung
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i formazione di scopazzi

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g Ertragsminderung
f diminution du rendement
i minor produzione, riduzione di raccolto

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g Zickzackwuchs
i croissance en zigzag
f sviluppo a zig zag

II. Dutch index with english equivalents

(acronecrose) zie *topnecrose*

acute fase, shockfase
acute phase, shock phase

aftakeling
decline

anthocyaanvorming
anthocyanin formation

antholyse, bloemoplossing
antholysis

apostasie
apostasis

atrofie
atrophy

(aucubabont)
zie *(aucubamozaïek)*

(aucubamozaïek), (aucubabont)
(aucuba mosaic)

---

bastnecrose, baststerfte
canker, bark necrosis

baststerfte zie *bastnecrose*

bladkrulling zie *krulling*

bladrandchlorose
zie *randchlorose*

bladrandvergeling
zie *randvergeling*

bladrolling
leaf rolling

bladval
leaf dropping, leaf casting, leaf abscission, defoliation

blastomanie
zie *heksenbezemgroei*

bloemdoorgroei zie *prolificatie*

bloemkleurbreking
breaking of flower colours

bloemoplossing zie *antholyse*

bontheid
variegation

bovengrondse knolvorming
aerial tuber formation

bronskleuring
bronzing

(calico)
(calico)

callosevorming, (overmatige) –
(excessive) callose formation

celinsluitsels
inclusion bodies, (viroplasts)

chlorose
chlorosis

chronische fase
chronic phase

cladomanie
zie *heksenbezemgroei*

---

diepnervigheid
rugosity

distorsie zie *verwringing*

draadbladigheid, naaldbladigheid
shoe stringing, (lacing)

draadspruitigheid
spindle-sprouting

dubbele knopen
double nodes

dwerggroei
dwarfing, stunting

eikebladpatroon
oak-leaf pattern

enatie, uitwas
enation

epinastie
epinasty

etsing
etching

figuurbont
line pattern

floëemnecrose
phloem necrosis

fyllodie
phyllody

(geelnervigheid)
zie *nerfvergeling*

gevlektheid zie *vlekking*

gevoeligheid
sensitivity

gezwel zie *tumor*

gomvorming
gummosis

groeiremming
growth reduction

heksenbezemgroei, blastomanie, cladomanie
witches' broom growth, blastomania, cladomania

heksenbezemverschijnselen
witches' broom phenomena

heksenbezemvorming
witches' broom formation

herstel
recovery

histoïde misvorming
zie *misvorming*

hyperplasie
hyperplasia

hypertrofie
hypertrophy

hypoplasia
hypoplasia

immuniteit, onvatbaarheid
immunity

inapparentie, symptoomloosheid, inapparente infectie, symptoomloze infectie
inapparency, inapparent infection

inapparente infectie
zie *inapparentie*

incubatieperiode, incubatietijd
incubation period

incubatietijd
zie *incubatieperiode*

inenting zie *inoculatie*

infectie
infection

inoculatie, inenting
inoculation

internodiënverkorting, stengellidverkorting
internode shortening, shortening of internodes

kanker
cankerous tumour en tumerous canker

kiemrustverkorting
reduction of seed dormancy

kreuk(el)ing, rimpeling, (krinkeling)
crinkling, crumpling, wrinkling

kringerigheid, kringvorming
ring formation

kringvlekvorming
ringspotting

kringvorming zie *kringerigheid*

(krinkeling) zie *kreuk(el)ing*

krulling, bladkrulling
curling, leaf curling

kurkvorming
cork formation

---

latentie
latency

lesie
lesion

lokale lesie
local lesion

lokale symptomen
local symptoms

---

maskering
masking

misvorming
malformation, deformation

mozaïek
mosaic

---

naaldbladigheid
zie *draadbladigheid*

necrose
necrosis

negatieve geotropie, versterking der –
intensification of the negative geotropy

nerfbandmozaïek
vein banding

nerfchlorose
vein chlorosis

nerfglazigheid
vein clearing

nerfmozaïek
vein mosaic

nerfnecrose
veinal necrosis

nerfvergeling, (geelnervigheid)
vein yellowing

---

olievlek
oil fleck

onvatbaarheid zie *immuniteit*

onvolledige verhouting
defective lignification

oogstreductie
zie *opbrengstdaling*

opbrengstdaling, oogstreductie
yield reduction

organoïde misvorming
zie *misvorming*

overgevoeligheid
hypersensitivity

---

paarskleurig
purpling

primaire symptomen
primary symptoms

(proliferatie) zie *woekering*

prolificatie, (bloemdoorgroei)
prolification

---

randchlorose, bladrandchlorose
edge chlorosis

randvergeling, bladrandvergeling
edge yellowing

resistentie
resistance

rimpeling zie *kreuk(el)ing*

roodkleuring
reddening

rotting
rotting

rozetvorming
rosetting

'rubberhout'
'rubbery wood'

ruwschilligheid
'rough skin'

---

scheutzwelling, takzwelling
shoot swelling

schubbigheid
bark scaling

secondaire symptomen
secondary symptoms

shockfase zie *acute fase*

smalbladigheid
leaf narrowing

spikkeling, stippeling
speck(l)ing, stippling, dotting

(spreidsymptomen)
zie *systemische symptomen*

stengellidverkorting
zie *internodiënverkorting*

steriliteit
sterility

stervormige barsten, vorming van –
star cracking

stervormige vlekking
asteroid spotting

stippeling zie *spikkeling*

streping
streaking, striping

symptoom, ziektesymptoom, ziekteverschijnsel
symptom, disease symptom

symptoomloosheid
zie *inapparentie*

symptoomloze infectie
zie *inapparentie*

syndroom, ziektebeeld
syndrome

systemische symptomen, (spreidsymptomen)
systemic symptoms

---

takafplatting
flattening of branches

takgeribdheid
(Duits: Astrilligkeit)

takzwelling zie *scheutzwelling*

(teratomata)
(teratomata)

thyllenvorming, overmatige –
excessive tylose formation

tolerantie
tolerance

topnecrose, topsterfte, (acronecrose)
top necrosis

topsterfte zie *topnecrose*

tumor, gezwel
tumour, (Am. tumor)

tussennervig mozaïek
interveinal mosaic

(uitdroging) zie *verdroging*

uitwas zie *enatie*

---

vatbaarheid
susceptibility

verbleking, witkleuring
blanching, bleaching

verbruining
browning

verdorring
withering

verdroging, (uitdroging)
desiccation

vergeling
yellowing

vergroening, virescentie
greening, virescence

verstoorde periodiciteit
disturbed periodicity

verwelking
wilting

verwringing, distorsie
distortion

virescentie zie *vergroening*

vlekkerigheid
mottling, (mottle)

vlekking, gevlektheid
flecking, spotting

---

waaierbladvorming
fan-leaf formation

watergebrek zie *watertekort*

watertekort, watergebrek
water deficiency, water shortage

witkleuring zie *verbleking*

woekering, (proliferatie)
proliferation

---

zetmeelophoping
accumulation of starch

ziekte
disease

ziektebeeld zie *syndroom*

ziektesymptoom zie *symptoom*

ziekteverschijnsel
zie *symptoom*

zigzag-groei
zigzag growth

zwartkleuring
blackening

III. German index with english equivalents

Abkürzung der Keimruhe
reduction of seed dormancy

Abzehrung, Verfall
decline

Adernaufhellung
vein clearing

Adernbänderung, Adernbandmosaik
vein banding

Adernbandmosaik
s. *Adernbänderung*

Adernchlorose
vein chlorosis

Adernmosaik
vein mosaic

Adernnekrose, ('Rippenbräune')
veinal necrosis

Adernvergilbung
vein yellowing

(Akronekrose) s. *Spitzennekrose*

Ätzmuster s. *Ätzung*

Ätzung, Ätzmuster
etching

akute Phase, Schockphase
acute phase, shock phase

Anfälligkeit
susceptibility

Antholyse
antholysis

Anthozyanbildung
anthocyanin formation

Apostasie
apostasis

Astrilligkeit
??????

Atrophie
atrophy

(Aucubamosaik)
(aucuba mosaic)

(Austrocknung) s. *Vertrocknung*

Auswuchs s. *Enation*

Beimpfung s. *Impfung*

Bildungsabweichung
s. *Missbildung*

Blastomanie
s. *Hexenbesenwuchs*

Blattfall
leaf dropping, leaf casting, leaf abscission, defoliation

Blattrandchlorose
s. *Randchlorose*

Blattrandvergilbung
s. *Randvergilbung*

Blattrollung
leaf rolling

('Bleiche') s. *Bleichung*

Bleichung, ('Bleiche')
blanching, bleaching

Blütendurchwuchs
s. *Prolifikation*

Blütenfarbenbrechung, (Buntstreifigkeit)
breaking of flower colours

('Bräune') s. *Braunfärbung*

Braunfärbung, ('Bräune')
browning

Bronzefärbung
bronzing

Buntblättrigkeit
variegation

(Buntstreifigkeit)
s. *Blütenfarbenbrechung*

Chlorose
chlorosis

chronische Phase
chronic phase

Deformierung s. *Missbildung*

diffuse Fleckung?
mottling, (mottle)

Doppelknoten, (Kurzknoten)
double nodes

('Dürre') s. *Verdorrung*

Eichenblattmuster
oak-leaf pattern

Einschlusskörper
inclusion bodies, (viroplasts)

(Einzelherd) s. *Lokalläsion*

Empfindlichkeit
sensitivity

Enation, Auswuchs
enation

Epinastie
epinasty

Erholung
recovery

Ertragsminderung
yield reduction

Fadenblättrigkeit
shoe stringing, (lacing)

Fadenkeimigkeit
spindle-sprouting, (hair sprouting)

Fächerblattbildung
fan-leaf formation

'Fäule'
rotting

Flachästigkeit, Zweigabplattung
flattening of branches

Fleckenbildung s. *Fleckung*

Fleckung, Fleckenbildung
flecking, spotting

Folgesymptome
s. *Sekundärsymptome*

(Gelbchlorose) s. *Vergilbung*

gestörte Periodizität
disturbed periodicity

Gummibildung s. *Gummosis*

'Gummiholz'
'rubbery wood'

Gummosis, Gummibildung
gummosis

Hexenbesenbildung
witches' broom formation

Hexenbesenerscheinungen
witches' broom phenomena

Hexenbesenwachstum
s. *Hexenbesenwuchs*

Hexenbesenwuchs, Blastomanie, Hexenbesenwachstum, Kladomanie, Triebsucht
witches' broom growth, blastomania, cladomania

Hyperplasie
hyperplasia

Hypertrophie
hypertrophy

Hypoplasie
hypoplasia

Immunität
immunity

Impfung (Beimpfung eines Blattes, **Verimpfung** eines infektiösen Saftes)
inoculation

inapparente Infektion
s. *Inapparenz*

Inapparenz, inapparente Infektion
inapparency, inapparent infection

Infektion
infection

(Initialsymptome)
s. *Primärsymptome*

Inkubationszeit
incubation period

Internodienverkürzung
internode shortening, shortening of internodes

(Kaliko)
(calico)

Kallosebildung, (übermässige) –
(excessive) callose formation

Kladomanie s. *Hexenbesenwuchs*

Korkbildung
cork formation

Kräuselung, leichte Kräuselung
crinkling, crumpling, wrinkling

Kräuselung, schwere Kräuselung
curling, leaf curling

Krankheit
disease

Krankheitsbild s. *Syndrom*

Krankheitssymptom
s. *Symptom*

Krebs
cankerous tumour und tumorous canker

Kringelbildung, Ringbildung, Pfropfenbildung (bei Kartoffelknollen)
ring formation

Kümmerwuchs s. *Zwergwuchs*

(Kurzknoten) s. *Doppelknoten*

Läsion
lesion

Latenz
latency

Linien- und Kurvenmuster
line pattern

(Lokalherd) s. *Lokalläsion*

Lokalläsion, (Einzelherd), (Lokalherd)
local lesion

Lokalsymptomen
local symptoms

Luftknollenbildung
aerial tuber formation

Maskierung
masking

Missbildung, Bildungsabweichung, Deformierung
malformation, deformation

Mosaik
mosaic

Nanismus s. *Zwergwuchs*

negative(n) Geotropie, Verstärkung der –
intensification of negative geotropy

Nekrose
necrosis

Ölfleck
oil fleck

Pfropfenbildung (bei Kartoffelknollen)
s. *Kringelbildung*

Phloemnekrose
phloem necrosis

Phyllodie
phyllody

Primärsymptome, (Initialsymptome)
primary symptoms

Prolifikation, (Blütendurchwuchs)
prolification

Purpurfärbung
purpling

Randchlorose, Blattrandchlorose
edge chlorosis

Randvergilbung, Blattrandvergilbung
edge yellowing

Rauhschaligkeit
'rough skin'

Resistenz, Widerstandsfähigkeit
resistance

Rindenbrand
canker, bark necrosis

Ringbildung s. *Kringelbildung*

Ringfleckenbildung
s. *Ringfleckung*

Ringfleckigkeit s. *Ringfleckung*

Ringfleckung, Ringfleckenbildung, Ringfleckigkeit
ringspotting

('Rippenbräune')
s. *Adernnekrose*

('Röte') s. *Rotfärbung*

Rosettenbildung
rosetting

Rotfärbung, ('Röte')
reddening

Schmalblättrigkeit
leaf narrowing

Schockphase s. *akute Phase*

Schuppigkeit
bark scaling

Schwarzfärbung
blackening

Sekundärsymptome, Folgesymptome
secundary symptoms

Spitzennekrose, (Akronekrose)
top necrosis

Sprenkelung, Tüpfelung
speck(l)ing, stippling, dotting

Stärkeschoppung
s. *Stärkestauung*

Stärkestauung, Stärkeschoppung
accumulation of starch

Stauchung s. *Zwergwuchs*

Sterilität
sterility

Sternfleckung
asteroid spotting

Sternrissigkeit
star cracking

Streifung s. *Strichelung*

Strichelung, Streifung
streaking, striping

Symptom, Krankheitssymptom
symptom, disease symptom

Syndrom, Krankheitsbild
syndrome

systemische Symptome
systemic symptoms

(Teratomata)
(teratomata)

Thyllenbildung, übermässige –
(excessive) tylose formation

Tiefadrigkeit
rugosity

Toleranz
tolerance

Triebschwellung
shoot swelling

Triebsucht s. *Hexenbesenwuchs*

Tüpfelung s. *Sprenkelung*

Tumor
tumour. (Am. tumor)

Überempfindlichkeit
hypersensitivity

unvollständige Verholzung
defective lignification

Verdorrung, ('Dürre')
withering

Verdrehung, (Verkrümmung)
distortion

Verfall s. *Abzehrung*

Vergilbung, (Gelbchlorose)
yellowing

Vergrünung, Vireszens
greening, virescence

Verimpfung s. *Impfung*

Verkrümmung s. *Verdrehung*

Vertrocknung, (Austrocknung)
desiccation

Verwelken, Welken, ('Welke')
wilting

Verzwergung s. *Zwergwuchs*

Vireszens s. *Vergrünung*

Wachstumshemmung
growth reduction

Wasserdefizit
water deficiency, water shortage

('Welke') s. *Verwelken*

Welken s. *Verwelken*

Widerstandsfähigkeit
s. *Resistenz*

Wucherung
proliferation

Zickzackwuchs
zigzag growth

Zweigabplattung
s. *Flachästigkeit*

Zwergwuchs, Kümmerwuchs, Nanismus, Stauchung, Verzwergung
dwarfing, stunting

zwischennerviges Mosaik
interveinal mosaic

IV. French index with english equivalents

abscission des feuilles
leaf dropping, leaf casting
leaf abscission, defoliation

accumulation d'amidon
accumulation of starch

altération de la périodicité
disturbed periodicity

anneaux, formation d' –
voir *cernes, formation de –*

anthocyanose
anthocyanin formation

antholyse
antholysis

aplatissement des branches
flattening of branches

apostasis
apostasis

arabesques
line pattern

atrophie
atrophy

---

balais de sorcière, croissance en –
voir *blastomanie*

balais de sorcière, formation de –
witches broom formation

balais de sorcière, phénomènes des –
witches' broom phenomena

blanchiment
blanching, bleaching

blastomanie, croissance de balais de sorcière
witches' broom growth,
blastomania, cladomania

'bois caoutchouc',
voir *'bois souple'*

'bois souple', 'bois caoutchouc'
'rubbery wood'

bronzage
bronzing

brunissement
browning

(calicot)
(calico)

callose, formation (excessive) de –
(excessive) callose formation

cernes, formation de – formation d'anneaux
ring formation

chancre
cankerous tumour et
tumorous canker

chlorose
chlorosis

chlorose des nervures
vein chlorosis

chlorose du bord de la feuille
edge chlorosis

coloration pourpre
purpling

'court-noué'
internode shortening,
shortening of internodes

craquelure en étoile
star cracking

croissance en zigzag
zigzag growth

---

déficience en eau, manque d'eau
water deficiency, water shortage

dépérissement
decline

desquamation corticale
bark scaling

dessèchement
desiccation et withering

diminution du rendement
yield reduction

distorsion
distortion

doubles noeuds
double nodes

éclaircissement des nervures
vein clearing

énation
enation

enroulement foliaire
leaf rolling

épinastie
epinasty

état latent, voir *latence*

---

feuille en éventail, formation de la –
fan-leaf formation

feuilles filiformes, formation de –
shoe stringing, (lacing)

filosité
spindle-sprouting, (hair sprouting)

flétrissement
wilting

frisolée faible, gaufrage
crinkling, crumpling, wrinkling

frisolée grave
curling, leaf curling

---

gaufrage, voir *frisolée faible* et *rugosité*

géotropisme négatif, intensification du –
intensification of negative geotropy

gravure
etching

gummose
gummosis

---

hyperplasie
hyperplasia

hypersensibilité
hypersensitivity

hypertrophie
hypertrophy

hypoplasie
hypoplasia

---

immunité
immunity

inclusions
inclusion bodies, (viroplasts)

infection
infection

infection inapparente
inapparency, inapparent infection

inoculation
inoculation

---

jaunissement
yellowing

jaunissement des nervures
vein yellowing

jaunissement du bord de la feuille
edge yellowing

---

latence, état latent
latency

lésion
lesion

lésion locale
local lesion

lignification défectueuse
defective lignification

liseré des nervures
vein banding

---

maladie
disease

malformation
malformation, deformation

manque d'eau,
voir *déficience en eau*

marbrure
mottling, (mottle)

masquage
masking

mosaïque
mosaic

(mosaïque aucuba)
(aucuba mosaic)

mosaïque des nervures
vein mosaic

mosaïque internervale
interveinal mosaic

moucheture
speck(l)ing, stippling, dotting

---

nanisme
dwarfing, stunting

nécrose
necrosis

nécrose apicale
top necrosis

nécrose corticale
canker, bark necrosis

nécrose des nervures
veinal necrosis

nécrose du liber
phloem necrosis

noircissement
blackening

---

panachure de la fleur
breaking of flower colours

peau rugueuse
'rough skin'

période d'incubation
incubation period

phase aiguë
acute phase, shock phase

phase chronique
chronic phase

phyllodie
phyllody

pourriture
rotting

prolifération
proliferation

prolification
prolification

---

réduction de la croissance
growth reduction

réduction de la durée du repos germinatif
reduction of seed dormancy

renflement des branches
shoot swelling

résistance
resistance

rétablissement
recovery

rétrécissement du limbe
leaf narrowing

rosettes, formation de –
rosetting

rougissement
reddening

rugosité, gaufrage
rugosity

---

sensibilité
sensitivity

stérilité
sterility

striure
streaking, striping

subérisation
cork formation

susceptibilité
susceptibility

symptôme (de maladie)
symptom, disease symptom

symptômes généralisés
systemic symptoms

symptômes localisés, symptômes locaux
local symptoms

symptômes primaires
primary symptoms

symptômes secondaires
secondary symptoms

syndrome
syndrome

---

tache huileuse
oil fleck

taches annulaires, formation de –
ringspotting

taches digitées
oak-leaf pattern

taches étoilées
asteroid spotting

tacheture
flecking, spotting

(tératomes)
(teratomata)

thylles, formation excessive de –
(excessive) tylose formation

tolérance
tolerance

tubercules aériens, formation de –
aerial tuber formation

tumeur
tumour, (Am. tumor)

---

variégation
variegation

virescence
greening, virescence

V. Italian index with english equivalents

accartocciamento, arrotolamento della foglia
leaf rolling

accorciamento degli internodi
internode shortening, shortening of internodes

accumulo di amido
accumulation of starch

alterazione del periodismo
disturbed periodicity

annerimento
blackening

antociani, formazione di –
anthocyanin formation

antolisi
antholysis

anulatura
ring formation

apostasi
apostasis

appassimento
wilting

appiattimento dei rami
flattening of branches

arricciamento grave
curling, leaf curling

arricciamento leggero
crinkling, crumpling, wrinkling

arrossamento
reddening

arrotolamento della foglia
vide *accartocciamento*

atrofia
atrophy

avvizzimento
withering

---

bandatura nervale
vein banding

blastomania, cladomania, sviluppo di scopazzi
witches' broom growth, blastomania, cladomania

bronzatura
bronzing

buccia ruvida
'rough skin'

---

caduta di foglie vide *filloptosi*

(calico)
(calico)

callosi, formazione (eccessiva) di –
(excessive) callose formation

cancro
cankerous tumour ed tumorous canker

cladomania vide *blastomania*

clorosi
chlorosis

clorosi delle nervature
vein chlorosis

clorosi marginale
edge chlorosis

---

deficienza idrica
vide *insufficienza idrica*

deformazione
vide *malformazione*

deperimento
decline

desquamazione della corteccia
bark scaling

disseccamento
desiccation

distorsione
distortion

---

enazione, escrescenza
enation

epinastia
epinasty

escrescenza vide *enazione*

'etching'
etching

---

fase acuta
acute phase, shock phase

fase cronica
chronic phase

fenomeni di cladomania ed antolisi
witches' broom phenomena

fillodia
phyllody

filloptosi, caduta di foglie
leaf dropping, leaf casting, leaf abscission, defoliation

foglie a 'ventaglio', formazione di –
fan-leaf formation

foglie filiformi, formazione di –
shoe stringing, (lacing)

(forme teratologiche)
(teratomata)

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geotropismo negativo, accentuazione del –
intensification of negative geotropy

germogli filanti, formazione di –
spindle-sprouting, (hair sprouting)

gommosi
gummosis

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imbrunimento
browning

immunità
immunity

inclusioni cellulari
inclusion bodies, (viroplasts)

infezione
infection

infezione non manifesta
inapparency, inapparent infection

ingiallimento
yellowing

ingiallimento delle nervature
vein yellowing

ingiallimento marginale (della foglia)
edge yellowing

ingrossamento dei germogli (dei rami), rigonfiamento dei germogli (dei rami)
shoot swelling

inoculazione
inoculation

insufficienza idrica, deficienza idrica
water deficiency, water shortage

inverdimento, virescenza
greening, virescence

iperplasia
hyperplasia

ipersensibilità
hypersensitivity

ipertrofia
hypertrophy

ipoplasia
hypoplasia

latenza
latency

'legno di caucciù'
vide *'legno di gomma'*

'legno di gomma', 'legno di caucciù'
'rubbery wood'

lesione
lesion

lesione locale
local lesion

lignificazione incompleta
defective lignification

lineolatura
line pattern

macchia d'olio
oil fleck

macchiettatura
vide *punteggiatura*

macchiettatura asteroide
asteroid spotting

maculatura
flecking, spotting

maculatura anulare
ringspotting

maculatura a foglia di quercia
oak-leaf pattern

maculatura diffusa
vide *mosaicatura (alquanto sfumata)*

malattia
disease

malformazione, deformazione
malformation, deformation

marcescenza vide *marciume*

marciume, marcescenza
rotting

mascheramento
masking

minor produzione, riduzione di raccolto
yield reduction

mosaicatura (alquanto sfumata), maculatura diffusa
mottling, (mottle)

mosaico
mosaic

(mosaico aucuba)
(acuba mosaic)

mosaico internervale
interveinal mosaic

mosaico nervale
vein mosaic

nanismo
dwarfing, stunting

necrosi
necrosis

necrosi apicale
top necrosis

necrosi corticale
canker, bark necrosis

necrosi del floema
phloem necrosis

necrosi delle nervature
veinal necrosis

nodi doppi
double nodes

periodo d'incubazione
incubation period

proliferazione
proliferation

prolificazione
prolification

punteggiatura, puntulatura, macchiettatura
speck(l)ing, stippling, dotting

puntulatura vide *punteggiatura*

purpurescenza
purpling

resistenza
resistance

restringimento della foglia, stenofillia
leaf narrowing

riduzione della quiescenza dei semi
reduction of seed dormancy

riduzione di sviluppo
growth reduction

riduzione di raccolto
vide *minor produzione*

rigonfiamento dei germogli (dei rami) vide *ingrossamento dei germogli (dei rami)*

ripresa
recovery

rosetta, sviluppo a –
rosetting

rottura del colore dei fiori
breaking of flower colours

rugosità
rugosity

sbiancatura
blanching, bleaching

schiarimento delle nervature
vein clearing

scopazzi, formazione di –
witches' broom formation

scopazzi, sviluppo di –
vide *blastomania*

sensibilità, sensitività
sensitivity

sensitività vide *sensibilità*

sindrome
syndrome

sintomi locali
local symptoms

sintomi primari
primary symptoms

sintomi secondari
secondary symptoms

sintomi sistemici
systemic symptoms

***sintomo* (della malattia)**
symptom, disease symptom

spaccatura stellata, spaccatura stellare
star cracking

stenofillia vide *restringimento della foglia*

sterilità
sterility

striatura
streaking, striping

suberosi
cork formation

suscettibilità vide *suscettività*

suscettività, suscettibilità
susceptibility

sviluppo a zig zag
zigzag growth

tilli, eccessiva formazione di - vide *tillosi*

tillosi, eccessiva formazione di tilli
(excessive) tylose formation

tolleranza
tolerance

tuberi aerei, formazione di –
aerial tuber formation

tumore
tumour, (Am. tumor)

variegatura
variegation

virescenza vide *inverdimento*