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S Y M P T O M S

OF VIRUS DISEASES IN PLANTS

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INTRODUCTION

Among plant pathogens, viruses occupy a particular place. In contrast with the parasitic organisms they can enter the plant in a passive way only through extremely small wounds. These may be produced by e.g. breaking of epidermal hairs, such as by mutual contact of diseased and healthy plants, or by insects. In this way they reach the living plant cell. There the viruses, being biochemical units themselves, take part in the metabolism of the plant in one way or another inducing biochemical changes. How this occurs is still unknown. These internal, invisible biochemical changes may lead to the production of several visible abnormalities. Due to their biochemical origin they are initiated at a cytological level, and subsequently may cause macroscopical alterations. Since the plant or plant parts suffer from them, the changes are known as disease symptoms or pathological phenomena (pathos = suffering). The whole group of symptoms caused by a given pathogen are known as the syndrome; they form the clinical picture of the disease.

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 or on, or in the neighbourhood of, the infected parts of the host. Therefore, in many cases they can be identified easily in contrast with virus diseases.

Thus, virus symptoms are products of a plant physiology upset by the virus. Obviously this host plant reaction depends on the physiological condition of the host. Since the condition depends largely on host species and variety, age, nutrition, climatic environment etc. it is easily understood 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, abnormalities due to toxic agents, overdosing of growth hormones used as weedkillers, or to some genetic disorders.

For these reasons virus diagnosis on the basis of symptoms, the so called clinical diagnosis, often is not reliable. However, in many cases, such as in the field, an aetiological diagnosis is impracticable. Since such a preliminary virus identification then can be based only on the symptoms observed, good knowledge of symptoms is indispensable.

In studying those viruses which are not (yet) sap transmissable in one or another way, symptomatology is an essential tool in diagnosis. Moreover in all laboratory tests with host plants knowledge of the plant reaction is needed.

Since in the rapidly increasing literature on plant virus diseases much confusion exists concerning the identity of the incitants, and as most diseases are named on the basis of characteristic symptoms, the proper use of terms is necessary. Therefore this paper aims at a critical survey, description and naming of virus symptoms in plants.

Before starting to describe the symptoms, however, it is essential to delimit the borderline between normal and abnormal plants, between healthy and diseased plants and between inapparent and apparent infection.

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. Many viruses have hosts in which infection always is invisible. In plant virology this permanent type of inapparency is called latency. These hosts are susceptible but insensitive or tolerant; they do not react in a visible way to the presence of virus. Since NISHIMURA in 1918 discovered this phenomenon, these hosts have been called carriers. The presence of virus, however, can be demonstrated by back inoculation on to sensitive hosts or by means of serology or electron microscopy. In inoculation experiments even a number of new viruses have been discovered when they produced symptoms in experimental hosts, such as the dodder latent mosaic virus (BENNETT, 1944), and the carnation latent virus (KASSANIS, 1954). The latter was discovered also to be latent in a number of potato varieties. 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. If the disease is masked more permanently it may be called recovery even though active virus is still present (BENNETT, 1955: recovery of Samolus parviflorus from curly top). The ultimate nature of this recovery phenomenon is unknown.

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 text-book on plant viruses of 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. So in the case of cereal and grass smuts e.g. BUTLER & JONES (1949) speak of a latent infection. Here infection becomes apparent when the ear or individual caryopses are developing at the end of the vegetative life of the host. Since "latent" literally means slumbering, 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 till the moment at which the presence of virus becomes apparent by the ability of the vector to infect healthy plants, often is called 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 has been suggested that the term "inapparent" can be used for all those 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 temporary absence of symptoms is needed, it will be difficult to get rid of this name. As it literally means unrecognizable but still visible, the term "masked" seems to be rather incorrect, however.

The concepts inapparent and latent 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 that 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 very little and fall beyond notice. Now the problem arises to distinguish between apparent infection having visible symptoms and inapparent infection being entirely imperceptible. For everyone who is acquainted with living nature it will be clear, however, 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. KUSTER, 1925; BLOCH, 1954).

This is demonstrated by the potato virus S, which was discovered serologically (DE BRUYN OUBOTER, 1952). The virus was studied thoroughly 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 show almost 100% infection. A few potato varieties appeared to produce a slight mosaic, whereas most varieties in an accurate comparison developed only very faint symptoms. 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 e.a. (1957) demonstrated how Ladino clover, after infection with a mixture of bean yellow mosaic virus and lucerne mosaic virus, even in cases where symptoms were scarcely noticeable, nevertheless often shows a decrease in yield. 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 with which the reaction of the plant is being 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 an incubation period during which physiological disorders are initiated, the first visible symptoms come into being. They may develop locally at the site of inoculation and are called local or primary symptoms. Here the host reaction is restricted to the infected cells and their immediate neighbourhood. These primary symptoms consist of a local discoloration, wilting or even necrosis of tissue. They are often called local lesions and will be described in detail in the next section. Rather often further invasion of the plant by the virus is prevented by this local reaction, especially when the infected cells become necrotic.

Usually the virus becomes systemic; it spreads internally 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, produce systemic or secondary 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.

It can happen that systemic infection also leads to the development of localized reactions.

This might be due to a low virus concentration in the transported phloem contents, giving rise to a localized systemic reaction on those spots where infectious units succeed in establishing multiplication centers.

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

So even the apical and basal parts of one and 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 originate most often in the top part of the leaf and mosaic and other symptoms in the lower part of the leaf if these leaves reached some differentiation before virus entry.

The difference in physiology of inoculated and systemically infected parts of the plant also may lead to a difference between the acute and the chronic phase. The acute phase may occur soon after inoculation and is more or less shock-like. This phase is characterized by severe symptoms leading to death of the host. Afterwards a chronic phase may occur, characterized by some recovery, when newly developing parts produce less severe symptoms. In one diseased plant acute and chronic phases even may interchange. A peculiar example is the "Echte Ackerbohnenmosaik"-virus in broad bean. In these plants the symptom expression shows a periodical course in severity. Groups of leaves with severe symptoms interchange with leaves with weak or no symptoms. The reason for the underlying interchange in virus concentration (PAUL & QUANTZ, 1959) is not yet known.

It has to be stressed here that virus symptoms are not restricted to young and growing plant parts as might be implied from the above text. ESAU (1948) already has pointed out that although young plant organs are relatively highly susceptible to virus infection, also fully formed plant parts may develop symptoms. The relatively low sensitivity of old 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.

Not only local symptoms may develop after inoculation, but systemic symptoms as well, if virus movement into such leaves is assured. Transport of assimilates to full grown leaves is almost excluded, however. 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. ESAU (1948) cites some examples in which symptoms in full grown plant parts develop.

NAMING OF SYMPTOMS

The word symptom is used to indicate the effects in the plant resulting from the presence of a pathogen. These effects may be named in terms indicating the products of a disturbed metabolism e.g. the "yellow edge" and the "mottle" of the leaf or the "rosette" at the extremities of the branches. The effects may also be named in terms indicating the processes underlying the altered appearance of the diseased plants e.g. "edge yellowing", "mottling", "rosetting". Both approaches lead to a different naming of symptoms: edge yellowing - yellow edge, mottle - mottling, rosette - rosetting.

Since in living material generally no static situations occur, the present author is inclined to prefer the use of the processes as a basis for naming symptoms. Moreover, the terms "yellow edge", "mottle", "rosette", "stunt", "wilt", "yellows" etc. are commonly used to indicate the diseases rather than the symptoms. Since the names of symptoms are code words more or less, they should be short, however. Therefore many authors prefer names as "mottle" and "wilt". Moreover, in some cases such as with "mosaic" no short term for the process underlying this abnormality exists. For these reasons both ways of naming symptoms are used in the literature on plant viruses, often in combination.

DESCRIPTION OF SYMPTOMS

Any organization of data should be based on a system. The same holds for a classification of virus symptoms. This will 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. After originating in biochemical changes the symptoms are initiated in the anatomy of the plant. Usually the deviations start at a cytological level. This makes the anatomical study of virus diseased plants of great importance. Since almost each virus symptom starts in the anatomy of the plant the anatomical disorders will not be described here separately. A survey of literature on anatomical aspects of plant virus disease problems has been given by ESAU (1938, 1948 and 1956). The most fundamental cytological alterations underlying virus symptoms are: depressive or destructive effects upon the chloroplasts, necrosis (death of cells), hypertrophy (increased size of cells), hyperplasia (increased number of cells) and hypoplasia (decreased number of cells). Frequently two or more of these types of reaction occur in combinations. The term hypertrophy is also used for abnormal enlargement of organs (cf. p. 26) and hypoplasia for abnormal reduction of organs, whereas the term atrophy is used for a complete reduction of organs. Since cork formation leads to a number of external abnormalities, this anatomical disorder will be discussed in a separate section.

As to the ontogenetic classification the deviations mentioned in groups I to V, viz. growth reduction, colour deviations, wilting and withering, necrosis, and visible biochemical changes, all are initiated at a cytological level. They find their origin in the abnormal cells themselves. (The succession of the groups I up to and including V is more or less arbitrary). Cork formation, mentioned in group VI, however, in itself is not abnormal. Abnormal is the way and site of initiation. It belongs more to organizational disturbances. This especially holds for the deviations mentioned in group VII, the malformations. They are especially due to a disorganization of usually normal cells. These disturbances are initiated at a histoid or at an organoid level. In group VIII a survey and discussion are given concerning the degree to which the virus particles themselves may contribute to the symptoms, directly or in forming inclusion bodies. The last group (IX) represents phenomena due to secondary causes introduced by a preceding virus infection.

Evidently nature can not be captured in a generally acceptable system. Therefore it will not be possible to classify virus symptoms with full satisfaction and to separate the groups of symptoms completely.

In the same way it appeared to be impossible to delimit the normal and the abnormal, the healthy and the pathological plant growth.

I Growth reduction

A great many viruses induce a non specific, general reduction in growth vigour (cf. also group IX). Such plants remain smaller in all their dimensions. When this is striking we speak of dwarfing or stunting. Morphologically, however, these plants are normal. This means that all the organs are reduced proportionally. This name has been applied improperly in some cases such as "Rubus stunt" for a witches' broom disease of raspberry (PRENTICE, 1950)(cf. also p.30).

When a plant acquires infection 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 e.a., 1959). (For rosetting, being a different phenomenon, cf. p.28).

Often the growth reduction leads to a reduction in size of the fruits. This sometimes may be very striking, such as in "little cherry" of sweet cherries, where the known symptoms are confined to the fruits. These have half or less than half normal size at picking time (FOSTER e.a., 1951). In connection with witches' broom growth rather commonly a reduction in size of leaves 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.30).

Growth reduction often occurs without accompanying symptoms. Rather usually, this reduction in gross plant size is not conspicuous, but the disease effect is noticed in a reduction in yield. This may be due to a reduction in size of the fruits, as was mentioned above, or to a reduction in total weight of the plants. Generally this is the most important economic aspect of virus diseases. Since size and yield of plants also depend on a number of other factors, such as nutrition, reductions in size and yield are generally difficult to recognize as virus symptoms. Moreover, especially here there is no borderline at all between normal and abnormal.

II Colour deviations

a Colour deviations in the leaves

Changes in colour of the leaves are quite common in virus diseases. They are mostly due to chlorophyll disorders, such as a delayed or a decreased chlorophyll production. That is why after some time mosaic symptoms may disappear more or less. Sometimes even a degeneration of chloroplasts may occur. This has been shown by ESAU (1944) in full grown leaves of beet infected with beet mosaic. Due to a lack of chlorophyll the presence of carotenes and xanthophylls becomes evident. As a result, the leaves as a whole or partly show a yellowing. In extreme cases the leaves or parts of the leaves even can become white. Besides the lack of chlorophyll e.g. in tobacco plants infected with tobacco mosaic virus an increased content of carotenes and xanthophyll (VENEKAMP, 1957) contributes to the yellowing phenomenon. Usually other anatomical disorders are also involved, such as a spherical shape of the palissade cells and a delayed formation of intercellular spaces in the mesophyll both leading to a decreased thickness of the yellow parts of the leaf. According to the survey of ESAU (1938, 1948) the picture differs with virus and host.

The yellowing can be general and then is named chlorosis, cf. the scientific name Chlorogenus callistephi H. for "aster yellows". Often, however for such diseases the name yellows is used e.g. "aster yellows, "sugar beet yellows", etc. It is evident that this complete yellowing only concerns the leaves developing after infection. This especially attracts attention after infection at a late stage of development of the plant. Then only the tips of the plants or of the branches show this yellowing e.g. "tip yellows" of peas (= pea leaf roll).

The yellowing also can be localized and be restricted to distinct areas of the leaf. In barley plants infected with the barley yellow dwarf virus the tips of the leaves are yellow. Usually, however, this yellowing subsequently proceeds to the entire leaf. The yellowing can also be confined to the edge of the leaf: edge yellowing, e.g. "strawberry yellow edge". Another type is the restriction of the discoloration to the veins, often together with some of the adjacent tissue. The pattern of vein yellowing is always very regular, however. A very nice example is "vein yellowing" of lucerne caused by the tip yellows virus of peas (VAN DER WANT & BOS, 1959).

Another example of a persistent yellow-vein virus is that causing "yellow-net" disease of sugar beet (SYLVESTER, 1948).

Related to this phenomenon is the symptom of vein clearing differing from vein yellowing, however, by showing clearing rather than yellowing of the veins. Moreover, this symptom is restricted to the veins. This name means that the veins become more or less translucent. This 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 and may be accompanied by chloroplast degeneration such as in curly-top beet leaves (ESAU, 1933). In many virus diseases vein clearing is a first or early symptom, usually being temporary, however.

> Some confusion exists in the literature as to whether the term vein clearing should be reserved for translucency of veins only or be extended to include a yellowing of some adjacent tissue. According to ESAU (1948) vein clearing should be considered equivalent to translucency of the veins. In the present author's opinion this is quite correct. The yellowing of the veins together with some adjacent tissue should be designated "vein yellowing". As will be described on p.11 a yellowing of the veins together with adjacent irregularly bordered leaf tissue is called "vein mosaic", whereas a colour change, dark or light, in bands along the main veins belongs to the phenomenon of "vein banding".

Under the term mosaic symptoms can be found a large group of colour deviations characterized by localized yellowing. In contrast to the above group here dark and light green or yellow parts of the leaf are irregularly variegated. Since MAYER introduced the name tobacco mosaic in 1886, mosaic symptoms are the first and best known group of virus symptoms. For a long time the names mosaic disease and plant virus disease were synonyms.

Many forms of mosaic can be distinguished. A very typical example of a uniform mosaic is the wellknown Abutilon mosaic (often named "Abutilon infectious variegation"), and "pea 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 straightlined. For a mosaic with only few very large yellow areas on the leaves the name Aucuba mosaic, referring to the variegated leaves of Aucuba japonica, sometimes is used e.g. "potato aucuba mosaic". The term calico, referring to brightly printed cotton dresses, has been used for a similar type of mosaic in which the brilliant yellow or sometimes almost white parts of the leaf dominate, such as in "potato calico" due to infection with the alfalfa mosaic virus (BLACK & PRICE, 1940) and in "peach calico" (BLODGETT, 1944).

In monocotyledonous plants with parallel veined leaves the light coloured parts have a tendency to become elongated. In this way a streak or stripe mosaic originates: e.g. "barley stripe mosaic" and "cocksfoot streak".

Based on the above definitions of chlorosis and mosaic the name "Abutilon infectious chlorosis", which is often used, is erroneous. "Abutilon mosaic" is preferable. The term "streak" e.g. in cocksfoot streak is confusing. The name does not indicate whether the streak is a mosaic or is necrotic (cf. p.17).

Other types of mosaic can be distinguished in which the mosaic patterns are confined to definite areas of the leaf. In vein mosaic the light-coloured parts are grouped along the main veins and these veins are included e.g. the "red clover vein mosaic". 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". If the mosaic patterns occur mainly between the main veins, the name interveinal mosaic is often used, e.g. in potato virus X. Sometimes the discoloration is restricted to sharply defined and brilliant green-yellow patterns formed by a single or multiple irregular lines or bands. This line patterning is characteristic for e.g. the "line pattern virus" in plum, peach and cherries (e.g. CATION e.a., 1951). In some cases the line pattern has the shape of an oak leaf.

In the light coloured parts of the leaf, growth is often impeded. Then the shape of the leaf also is abnormal e.g. in French bean infected with the common bean mosaic virus (Dutch: "rolmozaïek") (for more information cf. malformations).

In many cases the discoloration can be restricted to round flecks, dots, spots or speckles scattered all over the leaf. The phenomenon leading to these abnormalities is often called mottling. (In many publications, however, mosaic and mottle are used as equivalents). Sometimes they are the primary symptoms on the inoculated leaves. The margin of the spots may be sharp or diffuse. If in the latter case the entire fleck or the surrounding tissue is somewhat translucent they are named oilflecks. They are characteristic of the first symptoms of the Eckelrader disease or Pfeffingerkrankheit of sweet cherries (MULDER, 1951). Sometimes a light coloured ring surrounds a normally green centre. Also light and dark coloured concentric rings, often irregularly shaped, occur. Both are named ring spots. An important group of viruses is characterized by this type of symptoms viz. the ring-spot viruses (Annulus spp.).

In these spots necrosis often occurs (cf. necrosis).

A peculiar type of discoloration 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-diseased peach the foliage presents a striking depth and richness of green colour (HUTCHI 1933).

Entirely different from the chlorophyll disorders described above are those colour changes due to the abnormal formation of anthocyanins. These deviations from normal colour are often quite similar to mineral deficiencies. Some of the virus symptoms may be mentioned here. E.g. in clovers infected with the aster yellows virus a reddish-purple pigmentatio of the older leaves is a common phenomenon (HALISKY e.a., 1958). The barle yellow dwarf virus causes an intense orange-red coloration: "oat red leaf", "Blattröte oder Rotblättrigkeit" (e.g. RADEMACHER & SCHWARZ, 1958).

Bronzing, occurring in circular markings, as a network following the finer veins, or almost continuous over the affected portions of the leaves of tomatoes, is caused by tomato spotted wilt virus. The intensity of bronzing may vary from an almost imperceptible glaze only visible by turni the leaf to reflect light, to so deep a bronze as to be almost black. This phenomenon is due to a necrosis and collapse of epidermal cells overlying the still turgid, green, apparently healthy mesophyll tissue (SAMUEL e.a., 1930).

Brown and black coloration due to necrosis of tissues will not be described here (cf. necrosis). It is a rather common phenomenon in virus diseases and may be of diagnostic value, such as in "early browning" of peas (BOS, unpublished).

b Colour deviations in the stems

The stems can suffer from colour deviations similar to those found on leaves, since herbaceous stems are provided with similar chlorophyll conta ing tissues. E.g. in peach calico even the twigs become a 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, however. That is why they did not give rise to names of dis eases.

In potato "stem mottle" the name is inaccurate since only now and then do necrotic spots occur on the stems.

c Colour deviations in the 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 tulipbulbs during the years 1634-1637, when fortunes were paid for a single nicely broken tulip.

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: light breaking. It may give rise to very nice colour patterns (cf. the many colour pictures of VAN SLOGTEREN & DE BRUYN OUBOTER, 1941). In the case of pigment intensification small dark streaks or elongated flecks are developed: dark breaking. Both forms often occur together. These phenomena cannot be observed in white and yellow varieties since in their petals pigments are lacking in the epidermis. The disease is due to Tulipa-virus 1. Some types of light breaking may be of genetic origin. Dark breaking may also be due to rattle virus (VAN SLOGTEREN jr., 1958).

In several other plant species breaking of the flower colours is quite common and well known e.g. in gladiolus after infection with the bean yellow mosaic virus or the cucumber mosaic virus (KLINKOWSKI, 1956).

Besides these local colour changes in flowers general colour deviations also occur. The flower colours may be weakened, intensified or entirely changed. After infection with the Cucumis-virus 1 st.Chr., 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. Also here, yellow flowers seldom change and white flowers never do (NOORDAM, 1952).

Virescence or greening also 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 (cf. malformations, p. 30).

d Colour deviations in the fruits

Fruits also may show deviations in colour. Usually these are due to chlorophyll disorders. Principally these disorders are similar to those of leaves and stems. As beauty defects they influence the market value and may be of great economic importance. In plants with big fruits, colour changes attract special attention, such as in cucumber infected with the cucumber mosaic virus (e.g. TJALLINGII, 1952). This mosaic is also associated with deformations of the fruit (cf. p. 34).

III Wilting and withering

As a consequence of virus infection plants or plant parts may lose their turgidity and show wilting or even dry up and show withering. Mostly the latter process is a rapid one. Often wilting and withering occur together.

In peas, after mechanical inoculation, the white clover mosaic virus causes wilting and withering in the inoculated leaves. Under unfavourable circumstances the plants may entirely wilt ("pea wilt", BOS e.a., 1959). The loss of turgescence is well known in the leaves of onion plants infected with onion yellow dwarf virus.

As yet very little is known as to the exact origin of wilting and withering due to virus infection. Possibly the most important cause is lack of water due to an excess of transpiration or to a reduced supply of water e.g. due to necrosis in the vascular bundles. The reduced supply of water may also be due to a deposition of gum in vessels and other xylem cells and a precocious and excessive development of tyloses in the wood such as described for grapevine infected with the Pierce's disease virus (ESAU, 1948 b). This leads to a sudden wilting of vigorously growing young vines and the drying of grape leaves.

IV Necrosis

The local dying of cells or tissues, which is called necrosis, 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 always be observed.

Since location and type of necrosis is often characteristic, this symptom may be of diagnostic value. It may affect superficial cells or occur in deeper layers of tissue. It may involve 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, usually together with some surrounding cells, giving rise to local necrotic lesions. The mechanism of this local reaction is not quite 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 lead to 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 rot. This secondary phenomenon, however, is due to secondary fungi or, more often, to bacteria. They decay the dead material in a saprophytic way (cf. secondary phenomena). Under dry conditions the necrotic tissue may dry up.

a Necrosis in the leaf

Necrotic spots or stipples in the interveinal tissue may occur as a local result of the virus infection e.g. on leaves of Nicotiana glutinosa or Phaseolus vulgaris after mechanical inoculation with tobacco mosaic virus and a number of other viruses. Often these spots show concentric rings consisting of necrotic, yellow and dry tissue: ring spots (cf. also yellowing).

Sometimes the necrotic spots may be due to a systemic reaction. It is not known whether this is due to a low concentration of the virus resulting in a restricted number of places where infectious units become established. A nice example of systemic necrotic spots is "necrotic stipple" in store-cabbages due to the cauliflower mosaic virus (VAN HOOF, 1952).

When large numbers of necrotic lesions develop 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 early browning of peas. Usually the distribution is irregular but finally entire leaves may die. After having reached the veins and having been spread rather quickly within the veinal system of the leaf, the early browning virus of peas may induce a necrosis of these veins.

The same holds true for tobacco necrosis virus (stipple streak) in beans and the tobacco veinal necrosis virus (a strain of potato virus Y) in tobacco leaves. In this case, however, the veinal necrosis is confined to a restricted area around the point of inoculation. Leaves of pea, systemically infected with the early browning virus, may start with necrosis of the veins or of a part of the veins, but after some time the necrosis may proceed to the interveinal tissue.

b Necrosis in the stem

After having reached 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 in the leaves concerned.

An interesting type of vascular necrosis is "black root" in a number of snap bean varieties having field resistance to the common bean mosaic virus (cf. p.15). Then 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, pods etc.

Quite commonly a systemic reaction leads to death of young sprouts or tops of stems: top necrosis 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.

Concerning the origin and location of necrosis in the anatomy of stem different possibilities exist.

Especially in "phloem limited" viruses 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. 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).

As to the above mentioned systemic infection many types of necrosis are initiated in the phloem of the vascular bundles. In the black root disease of snap beans (cf. p.15), due to a systemic reaction of the bean yellow mosaic virus in hypersensitive varieties, necrosis affects not only the phloem, but also the cambium and the outermost layer of xylem (JENKINS 1941).

In the streak diseases, 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 two latter types of necrosis are manifest to the naked eye often as diffuse dark coloured streaks on stems, but also on petioles and main veins.

Stem necrosis also may find its origin in the parenchyma. In stems, but also in petioles and main veins, of French beans infected with the white clover mosaic virus parenchyma cells in the pericambium 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. 19) and is observable externally as dark greyish streak-like discolorations.

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 as well as the cortex shows considerable necrosis (BONING, 1931). Besides these internal necroses a stem can produce more superficial necrotic symptoms which are restricted more or less to the cortex. Presumably this necrosis resembles the necrotic lesions in leaves and petioles. E.g. 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).

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 on the areas enlarge and the necrosis may extend to the xylem. The necrosis may cause the bark to split. Frequently stems and branches are girdled, after which their upper portions die. (For the inaccurate application of the name canker in connection with this symptom, cf. p. 24).

Tubers of potatoes may show a series of necrotic phenomena.

In spraing or "corky ring spot", presumably due to a virus related to potato stem mottle (tobacco rattle), the cut surface of a tuber shows arc or ring like necrotic patterns, a ring spotting. Since the symptom is accompanied by some cork formation, this led to the name "corky ring spot". The German name "Pfropfenbildung" (formation of wads or pellets) expresses the three dimensional nature of the symptom.

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 differences 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. 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.

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).

c Necrosis in fruits and seeds

Necrosis may also occur in or on fruits. Pods of beans with stipple-streak virus (tobacco necrosis virus) often show necrotic ring patterns. The same holds for the pods of pea infected with early-browning virus. In the latter case the seeds touching necrotic parts of the pod wall may show necrotic dark brown spots as well. In these seeds necrosis even may penetrate into the cotyledons or sometimes into the germ (BOS, unpublished data).

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

V Biochemical changes, visible macroscopically or microscopically, in some instances with help of simple staining methods

Since viruses interfere with the physiology of the plant, all virus symptoms find their origin in biochemical changes. Of the processes involved only very little is known. From biochemical investigations, e.g. on tobacco mosaic diseased and healthy White Burley tobacco plants (VENEKAMP, 1957), however, we know that this virus induces, besides a decrease in chlorophyll content and an increase in the content of carotenes and xanthophylls, an evident increase in malic acid and a striking decrease in succinic acid content. How far these latter changes, being only detectable in an analytical way, will be of practical use as a symptom in diagnosis, will be difficult to predict. Moreover, since in normal plants biochemical composition (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 for virus infection. This has been demonstrated for sugar beets infected with yellows (JERMOLJEV & PRŮŠA, 1958) and for potatoes infected with virus X, virus Y, leaf roll virus and stolbur virus (JERMOLJEV, 1959 personal communication). A thorough discussion of these patho-physiological symptoms would go beyond the scope of this publication.

In a number of cases, however, biochemical changes are visible directly or after simple staining techniques by means of the microscope or even with the naked eye. In this case they may be of direct practical diagnostic importance. E.g. the white clover mosaic virus produces in French beans in petioles and stems, especially in groups of interfascicular parenchyma cells characteristic deposits of yellow brown to red brown gum-like substances (BOS, unpublished). This gummosis, however, is part of or is accompanied by necrosis (cf. p. 17). Externally these effects are visible as diffuse greyish internal discolorations. A very striking deposition of gum occurs in the xylem cells including the vessels of grape vine with "Pierce's disease" and of lucerne with "alfalfa dwarf disease", both being due to the same virus. Moreover, the xylem vessels of vine are occluded by a precocious and excessive production of tyloses. As a consequence afterwards growth reduction and wilting occur (ESAU, 1948 b).

In a number of diseases caused by "phloem limited" viruses, such as potato leaf roll, sugar beet yellows and pea leaf roll, abnormal accumulation of starch occurs in the leaves.

This can be easily demonstrated by means of iodine potassium-iodide after having removed the chlorophyll by means of alcohol. This accumulation of starch results in some thickness, stiffness and brittleness of diseased leaves.

For a long time the reduced transport of starch especially in potato was assumed to be due to the necrosis of the phloem (cf. necrosis). From the work of HENKE (1957), on sugar beet yellows, indications exist as to a disturbance of the transport of carbohydrates by a disturbed 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 even tubers of leaf roll diseased potatoes, preceding necrosis in these elements, attracts much practical and scientific attention. Whereas in phloem vessels of healthy plants the sieve plates are only covered with a thin layer of small plug of callose, the vessels of diseased plants show an increased quantity of callose. Even entire cells may be filled. This difference was first observed by VON BREHMER & ROCHLIN (1931). It is not yet known whether this abnormal callose production also is due to a disturbed activity of phosphatase. This callose can be easily stained with resorcin blue and 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 determining the abnormal callose production for diagnostic purposes was discovered somewhat simultaneously by IGEL & LANGE (unpublished but patented), BAERECKE (1955), HOFFERBERT & ZU PUTLITZ (1955), MOERICKE (1955) and SPRAU (1955).

A peculiar biochemical deviation is thedefective lignification of xylem and tracheids of apple, especially the variety Lord Lambourne, due to infection with the "rubbery-wood" virus. In cross-sections, 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, 1950 b). Older trees develop a "weeping" habit as the small branches bend under their own weight and under the weight of the crop.

VI Cork formation

The formation of cork is a normal phenomenon in plants. Especially in diseased plants it quite commonly occurs and often is due to wounding. Then it occurs secondarily. Cork formation is also often involved in virus diseases. Since no abnormal cells are produced but normal cells are incited to divide and form cork cells, an organizational disturbance is at hand in contrast to the previous symptom groups. In virus diseases cork formation has very seldom been the subject of serious anatomical research. So only little accurate information is available.

KLINKENBERG (1940) made some investigations on the anatomy of abnormal cork in the roots of Lupinus polyphyllus infected with "sore shin", presumably due to cucumber mosaic virus in her material. The cork occurs in a continuous layer at the bases of small intumescences on the roots, but 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 heavily 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 due to local growth retardation (VAN KATWIJK, 1955, 1956). In a presumably similar disease in the corky patches star cracking occurs. This phenomenon gave rise to the name "apple star-cracking virus" (JENKINS & STOREY, 1955).

VII Malformation

In the previous groups of virus symptoms the abnormalities found their origin in visible effects on the cell. They started at a cytological level. In a large group of virus diseases, however, the cells may be 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. Often external and internal structural abnormalities go hand in hand.

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

Malformations may be primary or secondary. In the first case the aberrations are the first visible virus symptoms. Secondary malformations are not evident until after the plant develops other symptoms such as localized yellowing, or necrosis which lead e.g. to leaf malformation. The differences will be discussed more in detail in the proper parts of this paper. The distinction between both types of malformations is not always sharp.

a) In the primary malformations the phenomena observed are the first visible deviations due to the virus infection. Presumably the disturbed correlation in development is due to a disturbed action of phytohormones (cf. also p. 33), such as a disturbed transport or distribution or 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 devided into two groups, in the same way that KUSTER (1911, 1925) did with the plant gall abnormalities produced by animals and fungi. The histoid deviations are due to an abnormal organization of distinct tissues or of tissues within a distinct organ. In organoid deviations the tissues and organs involved may be normal, whereas the organization of the organs, the relation between the organs, is abnormal. One has to realize that there exists no clear-cut borderline between the histoid and the organoid deviations. Especially in histoid growths cytological abnormalities are often involved. These aberrations of the cells then also may result from improper hormonal balance.

a 1) Since they do not produce normal organs the histoid deviations can not be described in terms borrowed from plant morphology. They are tumors, amorphous changes of histological nature, without any reasonable organizati

They have many features in common with human and animal cancers. That is one of the reasons why they attracted so much attention. Of course there are some differences due to differences in the ontogeny of animal and plant tissues (e.g. BLACK, 1952).

Opinions differ as to the correct name of the histoid deviations in plants caused by viruses. In view of their resemblance to histoid swellings caused by insects, especially the more voluminous ones, are often named galls, e.g. Galla fijiensis Holmes for the virus of Fiji disease of sugar cane. In a recent publication the cecidologist TROTTER (1954) even speaks of viro-cecids. The name gall must be considered inaccurate, however. Galls are swellings caused and inhabited by animal or plant parasites (KUSTER, 1925). So they are of critical ecological importance to the animal and plant parasite. This does not hold for the malformations due to virus infection, because the virus occurs in the other parts of the plants as well. Moreover, we have no evidence that the tumor is necessary to the survival of the virus. The most accurate name for the cancerous, wartlike swellings appears to be tumor. This term has already been used often in the literature (e.g. "wound-tumor" virus).

The origin and characters of tumors differ according to virus, host plant and part of the host. They are mostly composed of irregularly proliferating, badly organized tissue.

A number of viruses are able to induce tumors on the leaves. 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. They are due to hypertrophy (enlargement) and hyperplasis (increase in number) of palisade cells. Eventually the proliferating tissue protrudes through a crack in the epidermis. In the transverse direction only two or three palisade cells enlarge simultaneously at the beginning. In the longitudinal direction rows of cells of considerable length behave similarly, thus forming a ridge instead of a rounded projection (CALDWELL & JAMES, 1938). These swellings more or less resemble the so called intumescences, caused in many plant species by excessive humidity. The small elongated tumors on the under surface of the leaves of sugar cane infected with Fiji disease virus find their origin in abnormal proliferation of the phloem or tissues immediately adjoining the phloem (KUNKEL, 1924). This is why they extend along the under surface of the veins.

In crimson clover the wound-tumor virus induces irregular swellings of the veins("clover big vein") due to development of many small tumors in the phloem. In the leaf petiole, stem and root many of these tumors are only found internally, not showing on the surface as protuberances (LEE & BLACK, 1955).

Other peculiar protruding growths, being of more restricted size and proliferating less irregularly than tumors, are generally called histoid enations. They sometimes arise from the lower surface of the veins of crimson clover infected with wound-tumor virus. They also occur in lucerne, mostly only one per leaflet, after infection with a virus found in Rumania (BLATTNÝ, 1959). These enations are usually restricted to the main and side veins. They are the more protruding since they are placed on top of a local funnel shaped depression of the leaflet. The excrescences may vary in form from large or small papillae to short, whitish spines.

Well known and intensively studied are the tumors on stems and on the roots of sweet clover (Melilotus alba), and on the roots of sorrel (Rumex acetosa) and a number of other plants, due to the wound-tumor virus. This virus was not found originally in plants, but was discovered accidentally by BLACK (1945) in nymphs of the leafhopper Agalliopsis novella in the vicinity of Washington, D.C.. The stem tumors may attain a diameter of about 1 cm. The tumors are produced in systemically infected plants only after wounding (BLACK, 1946). A natural wounding occurs in roots producing side roots. These are initiated in the pericycle and mechanically break through the cortex. Tumors develop close to the wounded cells in the pericycle (LEE, 1955). According to this author tumors may be formed even at the bases of bacterial nodules.

In addition to tumors and enations more extensive swellings may develop. In the stems of sweet cherry, var. Napoleon, in Oregon U.S.A. "cherry black canker" virus produces slightly swollen areas. Later on these swellings split and grow into rough black cankers (ZELLER e.a.,1951). Prune diamond canker (SMITH & THOMAS, 1951) is a somewhat similar virus disease. This symptom has not been described in detail. Possibly secondary necrosis is involved. According to a discussion of literature of ZYCHA (1955) no accurate definition of the term canker is known. In plant pathology the English name canker primarily applies to localized necrosis in stems and twigs, leading to death of the bark up to the wood (cf. also p.17). Especially in "perennial cankers" (German "Krebs"). This induces the production of a callus around the wound.

This development may be different from that of the above diseases, both leading to a similar product, however. Therefore the term canker may be rather correct for these virus symptoms.

Even entire stems or shoots may demonstrate shoot swelling. A good example is the notorious "swollen shoot" disease of cacao in Western Africa (e.g. POSNETTE, 1947). Especially suckers arising from the base of the trunk may show pronounced swellings, amounting to twice the diameter of the normal stem. They may be nodal or internodal, 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 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. Due to one sided growth reductions, distortions of branches may occur. Sometimes spindly 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. In the same regions, the cortex, however, has more than twice the normal thickness (BLUMER, 1956).

Histoid outgrowths may even occur on fruits, such as in the peach "wart" disease (e.g. BLODGETT e.a. 1951). On young fruits bleached bumps or raised welts may develop on or near the stylar end, and 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 bonelike, but usually it is tough and leathery.

a 2) Within the organoid deviations 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 on their morphological significance was considered in a special branch of botany named teratology (cf. the textbooks of MASTERS, 1869, and PENZIG, 1921-1922).

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

Apparently the organoid deviations 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 due to virus infection are quite common. Most of them can be reduced to a disturbed relation in development of veinal and interveinal tissue. Often the growth of the laminar tissue is greatly reduced. This may lead to leaf narrowing, e.g. in cherries with Pfeffinger or Eckelrader disease. A very common example is tomato "narrow leaf" due to infection with tobacco mosaic virus or cucumber mosaic virus. Leaflets of infected tomatoes may get 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. TEPFER & CHESSIN (1959) studied the development and anatomy of narrow bladed and shoestring leaves of Turkish tobacco, Xanthi strain, after infection with tobacco mosaic virus. The shoestring 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 virus. 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 cause of previous deformities of the leaf even the veinal tissue may show a reduced growth rate in comparison with the interveinal tissue. This leads to a lumpy, bubbled surface of the leaf such as in tobacco leaf curl.

A last and very peculiar morphological aberration of the leaf is the abnormal enlargement or hypertrophy of stipules of apple due to the apple witches' broom virus. This symptom is of important diagnostic value.

Epinasty is a quite common phenomenon in virus infected plants, but may also be due to a number of other factors. It is caused by an excessive growth of the upper surface of the organ such as the petiole and the leaf blade. This leads to a downward curling of the entire leaf.

For a number of deformities due to secondary local growth reductions in the leaf cf. p.34 .

Enations already were mentioned in the group of histoid deviations. A number of enations on leaves exist, however, which have to be classified as organoid deviations, when they have the internal structure of a normal leaf: organoid enations. Usually they develop on the under-surface of the leaf. They are often associated with chlorotic areas, which they surround as fringes. They may consist merely of rough ridges, being of a more histoid nature. 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 at 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 enations on the leaves of Nicotiana glutinosa and Lycopersicum esculentum infected with the aspermy virus (a strain of the cucumber mosaic virus) have been studied recently by PRAGER (1958) for their morphology, anatomy and histogenesis. They 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) this virus produces enations even in the corolla of Petunia hybrida. The funnel- or cup-shaped enations caused by the leaf curl virus (kroepoek virus - Nicotiana-virus 10) 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 have their origin 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 infected with fan-leaf disease usually show short internodes, some of them often being entirely lacking. The latter phenomenon leads 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: rosetting. Some examples of virus diseases characterized by 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' brooming. However, this is an entirely different symptom (cf. p.29). Usually in rosette diseases the leaves involved also show some abnormalities.

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).

A very peculiar effect of a virus on the morphology of fruits is caused by the quercina virus (tobacco severe etch, Nicotiana-virus 7) on the fruits of thorn apple (Datura stramonium). The spines on the capsules are partially or entirely suppressed due to the virus infection (BLAKESLEE, 1921).

An extremely interesting group of organoid modifications due to viruses is formed by the witches' broom phenomena. They are caused by a complex of changes in vegetative and sexual parts of the plant influencing 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). This author gives a survey of an extensive number of witches' broom viruses producing these phenomena. Among them are listed virus diseases such as "aster yellows", "clover virescence", "tomato big bud", "stolbur", "rubus stunt" etc.

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 after a late infection during or at the beginning of flower initiation, floral abnormalities may prevail. This often led to confusion in the naming of these diseases. Inaccurate or confusing names and synonyms will be mentioned in the following text.

Due to an excessive and precocious development of buds leading to extravagant branching, the plant gets a bushy, brooming appearance. Since this systemic brooming is comparable to local brooming, caused by e.g. 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. The erect position of the newly formed branches, indicating an enhanced negative geotropy, contributes to the broom like habit. For this phenomenon the names blastomania and cladomania, adopted from teratology, referring to the mania, the rage to develop buds and branches, are very correct (The Greek word "blastos" means sprout but also sprouting or shooting, the word "klados" means 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, generally is 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 is generally used in biology for uncontrolled growths of a histoid nature. In this connection proliferating means sprouting in the sense of sprouting yeasts. This sprouting is fundamentally different from the excessive or abnormal sprouting of buds in witches' broom diseased plants, or the development of sprouts within flowers. The latter processes are of organoid nature. The terms "proliferation" and "prolification", derived from teratology, are commonly used only for the development of sprouts in flowers (see below). Both have the same meaning, the only difference being that prolification means the making of a sprout and proliferation the bearing of a sprout. Since in practice proliferation has been applied to histoid sprouting of tissue, the term prolification is preferred for organoid sprouting. Literally this term also could be used for excessive sprouting of vegetative buds. However, the terms blastomania or cladomania are quite correct for this phenomenon, since the development of axillary buds into vegetative sprouts in itself is not abnormal. Sprouting in excessive numbers is abnormal; this is a mania.

Woody plants may show a slow spread of virus. The infection may then be more or less local for a rather long time. The term witches' brooming more refers to this formation of local brooms, e.g. in the "brooming disease" of black locust (Robinia pseudo-acacia) (HARTLEY & HAASIS, 1929).

With infection at a late stage of development of the plants especially the extremities of the branches or inflorescences may produce local witches' brooms. So in Australia the witches' broom disease of lucerne sometimes has been called "bunchy top" (cf. EDWARDS, 1935). In the same way a witches' broom disease of peanut has been called "bunchy plant" (MORWOOD, 1954) and a similar disease of pecans "bunch disease" (COLE, 1937).

After infection at an early stage of development, a plant may remain very small and have the appearance of a dense, bushy broom.

This has often inaccurately been called "stunting", but is quite a different phenomenon (cf. stunting). This led to the confusing name "Rubus stunt" (PRENTICE, 1950) for a disease evidently belonging to the group of witches' broom virus diseases.

In witches' brooms the dense branching is usually accompanied by small leaf size. This symptom gave rise to the names "little leaf of brinjal" (THOMAS & KRISHNASWAMI, 1939) for a witches' broom disease of Solanum melongena in India and "legume little leaf" (HUTTON & GRYLLS, 1956) for a similar disease of subtropical pasture species in Australia. Since in diseases caused by other viruses small leaves are rather common, this name does not indicate a characteristic symptom of the diseases concerned.

Witches' broom viruses also disturb or completely prevent floral initiation. After this physiological disturbance, initiation and development, also of already partly differentiated flower buds, proceeds in an exclusively vegetative manner. As a consequence, antholysis develops, showing all intermediate stages between a normal flower and an entirely vegetative leafy branch. The ultimate result depends on the stage of flower initiation at the time of infection or more accurately 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 of normally white or coloured floral parts through the development of chloroplasts (cf. also colour deviations of flowers). This phenomenon is characteristic of such diseases as "virescence of tomato" (a synonym 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 in a later stage of antholysis the floral parts develop as more or less normal foliage leaves, the phenomenon is called phyllody.

In sepals this condition is often associated with considerable hypertrophy such as in flowers of raspberry infected with the Rubus witches' broom disease ("Rubus stunt"). In tomatoes infected with stolbur and tomato big bud virus the calyx is very much enlarged, even before opening of the flower. This leads to the "big (flower) bud" symptom. Stamens are only rather seldom phylloid, but they also may show interesting phyllody such as in Tropaeolum (BOS, 1957 a). 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. The ovules also are often present as small leaf-like organs at the edge of the open carpel. On this basis witches' broom virus diseases sometimes got named "phyllody", such as "phyllody of sun 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. Usually this only concerns the stipe or gynophore, which is the internode between the insertion of stamens and carpels. In this way 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) 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. 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. (For the differences between the terms prolification and proliferation cf. p. 29). The newly formed sprouts usually are vegetative, often much ramified, leafy branches. Especially in the case of central prolification a new inflorescence or flower may develop. 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. This 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 precocious 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 entirely fail to produce flowers. It is clear that all the witches' broom phenomena lead to complete sterility of the infected plants (cf. the name "sterility" of Sesamum indic in India (ROY, 1931). This characteristic makes the witches' broom virus diseases of particularly great economic importance. Infected plants do not produce valuable seeds nor fruits. For this reason such names as "diseases causing seedlessness in plants" (BOJŇANSKÝ & BLATTNÝ, 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" has been proposed (BLATTNÝ, 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 development of the plant at the time of infection[1]. After infection during vegetative development the witches' broom growth attracts most attention. 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, 1957 a) that the name "witches' broom virus disease" 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. This suggests a reduction of the seed dormancy. The intensification of negative geotropy due to virus, mentioned before, makes the normally positive geotropic stipes of peanut plants negative geotropic.

1) FRAZIER & POSNETTE (1957) claimed 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. The present author working with a similar virus in The Netherlands has been unable to obtain the same results.

Then, the stipes, having curled downward and even after having penetrated the soil and started pod formation, may curl upwards again. The same phenomenon can be observed in the pedicels of tomato plants infected with stolbur or tomato big bud. Instead of bending downwards the pedicels show an upright position. The same holds for the pedicels of cranberry infected with "false blossom" (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 virus (TODD, 1958) and with stolbur (BOJŇANSKÝ, 1958). Tubers of potato plants infected with stolbur virus (SAVULESCU & POP, 1956), potato witches' broom virus (TODD, 1958), "aster yellows" virus (LARSON, 1959), and perhaps with tomato big bud virus (WEBB & SCHULZ, 1958), after germination often show spindly sprouting or hair sprouting. They produce spindling, spindle 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.

The morphological disorders caused by this important group of viruses are reducible to a number of physiological disorders, viz.

1) abnormal intensification of the vegetative tendency,
2) suppression of the sexual tendency,
3) suppression of the apical dominance,
4) intensification of negative geotropy (even replacing positive geotropy),
5) reduction of the seed dormancy.

Since the processes involved are generally assumed to be hormone regulated, a hormonal disturbance caused by these viruses is suggested. (For more details cf. BOS, 1957).

b) The secondary malformations are due to other abnormalities which find their origin primarily in the biochemical changes due to 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 due to 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. The result is shown in the symptoms produced by the common bean mosaic virus in the leaves of French beans. 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 catterpiller-like. A similar phenomenon of deformation starting as a mosaic may be observed in fruits e.g. of cucumber after infection with cucumber mosaic virus (TJALLINGII, 1952). At the same time the margin of the leaf tends to curl or roll downward. This leaf rolling also may be upwards, such as in potato leaf roll. More irregular puckering of the blade commonly is called leaf curling or crinkling, such as in "sugar beet leaf curl" and "turnip crinkle". Leaf rolling and leaf curling are very common phenomena in virus diseases. Sometimes the entire venation may remain behind in comparison with the interveinal tissue. This gives the surface of the leaf a bubbled appearance e.g. in tobacco leaf curl. This phenomenon, however, belongs more to the primary malformations and is of organoid nature (p.26).

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

Evidently many malformations of leaves also may be due to primary, 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 necroses and excessive formation of sklerenchyma in stony pit of pear also leads to malformation.

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 delayed the detection of the nature of virus diseases for a long time. Without demonstrating 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 pathogens often may be easily recognized.

In 1939 KAUSCHE, PFANKUCH & RUSKA, however, discovered that the corpuscular nature of viruses, which already had been assumed on the basis of a number of observations, could be seen by means of an "Übermikroskop", an electron microscope with extremely high resolving power in comparison with the conventional light microscope. 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 are transmissible mechanically in one way or another.

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 first category, to 120 to 1250 mμ for the latter group. The width of these elongated particles varies from about 10 to 30 mμ.

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 grid for one to two seconds only. After drying, the preparation is ready for shadow casting and then for being studied in the electron microscope. With this easy method the present author already obtained excellent results, such as with the red clover vein-mosaic virus (HAGEDORN, BOS & VAN DER WANT, 1959). Since in the future new and improved techniques will be developed, undoubtedly the observation of virus particles in diseased 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 5-30 μ. In some diseases the morphology of these bodies may contribute to the identification of the virus.

Inclusion bodies have been known for a very long time. IWANOWSKI (1892, 1893) discovered two types of inclusions in the cytoplasm of mosaic diseased tobacco, viz. amorphous bodies, the so called X-bodies, and crystalline bodies. Both types of inclusions may occur independently, but changing of the first into the latter type has also been observed (KASSANIS & SHEFFIELD, 1941). The presence of bodies within the nucleus, intranuclear inclusions, is rather rare in plant viruses, in contrast to insect viruses. However, they do occur e.g. in tobacco with severe etch disease (KASSANIS, 1939).

The inclusion bodies can be stained in several ways a.o. with trypan blue. Especially the x-bodies can be easily mistaken for the nucleus. For some time the inclusions have been assumed to consist of virus protein (SHEFFIELD, 1946), whereas in x-bodies also other constituents may occur (SHEFFIELD, 1931). By means of the electron microscopy of ultra thin 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.

On the contrary, the crystalline inclusions show a regular crystalline arrangement of virus particles. How the virus particles aggregate to these structures is not yet known.

This makes clear how virus particles themselves may produce visible (by means of the microscope) deviations and how they corpuscularly 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.

IX Phenomena due to secondary causes

In virus diseased plants a general reduction in vitality often occurs. 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 alfalfa mosaic virus infected Ladino clover plants appeared to suffer more from frost than did healthy ones (ROBERTS, 1956).

Due to this weakening, virus diseased plants also may have an increased susceptibility to secondary infections with fungi and bacteria. E.g. peas with leafroll (tip yellows) often are infected with the soil inhabiting Fusarium solani. Until recently, this fungus was even assumed to be the incitant of the disease. This fungus, however, turned out to be unable to attack a healthy pea plant (HUBBELING, 1954). Broad bean plants infected with the same leafroll virus show an increased susceptibility to Botrytis fabae Sardiña, in comparison with healthy ones (TINSLEY, 1959). According to HEILING e.a. (1956) infection of sugar beets with yellows virus promotes infection with the fungus Cercospora beticola.

Rot 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 further decay of the dead tissue, called wet rot. In contrast, fungi usually produce a dry rot such as in stolbur infected 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.

INDEX WITH GERMAN AND DUTCH EQUIVALENTS

edge yellowing	Randvergilbung Blattrandvergilbung	randvergeling bladrandvergeling	
enation	Enation Auswuchs	enatie uitwas	
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fan leaf formation	Fächerblattbildung	waaierbladvorming	
flattening of branches	Flachästigkeit	takafplatting	
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ALPHABETISCHE LIJST VAN NEDERLANDSE NAMEN

MET KORTE DEFINITIES

Nederlands	Engels	Duits	Pag.
acute fase	acute phase	akute Phase	5

spoedig na infectie optredend deel van het ziekteverloop, dat gekenmerkt is door ernstige symptomen, die soms aanleiding geven tot de dood van de plant.

anthocyaanvorming	anthocyanin formation	Anthozyanbildung	12

abnormale roodkleuring ten gevolge van een versterkte vorming van anthocyaan.

antholyse, bloemoplossing	antholysis	Antholyse	30

het proces, waarbij allerlei tussenstadia, varierend van een normale bloem tot een geheel vegetatieve tak met bladeren, worden gevormd. Het resultaat van bloemoplossing is afhankelijk van het stadium van bloemaanleg op het moment van aantasting.

apostasie	apostasis	Apostasie	31

verlenging van de bloembodem ten gevolge van de ontwikkeling van één of meer internodiën; vaak betreft het alleen het lid tussen de inplanting van de meeldraden en de stamper(s), waardoor laatstgenoemd orgaan hoog boven de bloem kan uitsteken.

atrofie	atrophy	Atrophie	7

het niet tot ontwikkeling komen van weefsels en organen.

aucubamozaïek aucubabont	aucuba mosaic	Aucubamosaik	1

mozaïek of bontheid, waarbij de vaak zeer opvallende vergeling beperkt is tot enkele vrij grote vlekken op het overigens normaal groene blad.

bladkrulling	leaf curling crinkling	Blattkräuselung	33

onregelmatige kroesing en krulling van de bladschijf.

bladrandvergeling zie randvergeling			9
bladrolling	leaf rolling	Blattroll(en)	34

het naar beneden of naar boven ineenrollen of naar binnen buigen van de bladrand.

bloemkleurbreking	breaking of the flower colours	Blütenfarbenbrechung (Buntstreifigkeit)	13

streping of langwerpige vlekking van de kroon- of bloemdekbladeren in niet wit of geel gekleurde bloemen door een plaatselijk ontbreken van de kleurstof in de epidermis, waardoor de witte of gele kleur van het onderliggende weefsel zichtbaar wordt ("lichte breking") of een plaatselijke ophoping van kleurstof in de epidermis ("donkere breking").

bloemoplossing
zie antholyse — 30

bontheid
zie vlekking — 11

bronskleuring (bronsvlekken)	bronzing	Bronzefärbung (Bronzeflecken)	12

bronsachtige verkleuring doorgaans van normaliter groen gekleurde plantedelen.

callosevorming, overmatige -	callose formation, excessive -	Kallosebildung, übermässige -	20

vorming van buitensporige hoeveelheid callose op zeefplaten en in zeefvaten van stengels en aardappelknollen.

celinsluitsels	inclusion bodies	Einschlusskörper	36

abnormale lichaampjes in de cel, van amorfe of kristallijne aard, laatstgenoemde soms in de kern.

chlorose	chlorosis	Chlorose	9

bleekzucht of algemene vergeling.

chronische fase	chronic phase	chronische Phase	3

na de acute fase optredend deel van het ziekteverloop, dat gekenmerkt is door enig herstel van de plant waarbij aanhoudend milder symptomen optreden.

distorsie	distortion	Distortion	34

verwringing of verdraaiing van blad- of stengelorgaan.

draadbladigheid naaldbladigheid	shoe stringing	Fadenblättrigkeit	26

extreme vorm van smalbladigheid (zie aldaar), waarbij vrijwel, of in het geheel geen, bladmoes en alleen een hoofdnerf tot ontwikkeling komt.

draadspruitigheid	spindly sprouting hair sprouting	Fadenkeimigkeit	33

het kiemen van aardappelknollen met dunne, draadvormige spruiten.

dubbele knopen dubbelknopigheid	double nodes	Doppelknoten Kurzknoten	28

internodiënverkorting (zie aldaar), welke beperkt is tot enkele willekeurige leden, die nagenoeg geheel kunnen ontbreken, waardoor twee opeenvolgende knopen dicht opeen komen te zitten.

dwerggroei nanisme	dwarfing stunting	Zwergwuchs, Stauchung, Kümmerwuchs, Nanismus	8

het kleiner blijven van de plant in al haar afmetingen t.g.v. groeiremming (zie aldaar).

enatie uitwas	enation	Enation Auswuchs	24,2

uitgroeisel aan bladorganen, kan papilvormig en dan meestal klein zijn (histoide enatie) of bladachtig en dan meestal groter zijn (organoide enatie).

epinastie	epinasty	Epinastie	27

niet specifiek door virus veroorzaakte, benedenwaartse kromming van bladsteel en bladschijf.

figuurbont zie figuur mozaïek			11

figuurmozaïek figuurbont	line pattern	Linienmosaik, Bandmosaik	11

mozaïekpatroon, waarbij de verkleuring beperkt is tot helder groene tot gele lijnen of banden welke grillig over het bladoppervlak verlopen.

fyllodie	phyllody	Phyllodie	1

het loofbladachtig worden van bloemorganen, waarbij deze min of meer de vorm en soms afmetingen van normale loofbladeren aannemen.

geelnervigheid zie nerfvergeling			

gevlektheid zie vlekking			1

gomvorming	gummosis	Gummosis Gummibildung	19

vorming van gomachtige stoffen in cellen en intercellulairen, vaak samengaand met necrose.

groeiremming	growth reduction	Wachstumshemmung	8

algemene reductie in groei en ontwikkeling, aanleiding gevend tot dwerggroei (zie aldaar) en opbrengstdaling.

heksenbezemgroei blastomanie cladomanie (takzucht?)	witches' broom growth blastomania cladomania	Hexenbesenwuchs (- Wachstum) Blastomanie Kladomanie Triebsucht	29

het voortijdig en in buitensporige mate uitlopen van knoppen; door de sterke vertakking, waarbij de nieuwgevormde takken tevens een steile, opgerichte stand hebben, heeft de plant een bossig, heksenbezemachtig aanzien.

heksenbezemverschijnselen	witches' broom phenomena	Hexenbesenerscheinungen	28,

complex van afwijkingen bestaande uit heksenbezemgroei (zie aldaar), samengaande met bloemoplossingsverschijnselen (zie aldaar), waardoor de plant aan zijn takuiteinden en in de bloemgestellen heksenbezems vormt, of als geheel een heksenbezemachtige habitus krijgt.

heksenbezemvorming	witches' brooming	Hexenbesenbildung	2

de vorming van plaatselijke heksenbezems (in tegenstelling tot heksenbezemgroei, waarbij de gehele plant de groeiwijze en uiteindelijk de habitus van een heksenbezem verkrijgt).

herstel	recovery	Erholung	21 e.v.

het geheel of grotendeels verdwijnen van de symptomen voor de verdere levensduur van de plant.

histoïde misvorming zie misvorming			21 e.v.

hyperplasie	hyperplasia	Hyperplasie	7

abnormale toename van het aantal cellen.

hypertrofie	hypertrophy	Hypertrophie	7,26

abnormale vergroting van cellen, weefsels of organen.

hypoplasie	hypoplasia	Hypoplasie	7

abnormale afname van het aantal cellen en abnormale verkleining van organen.

inapparentie inapparente infectie	inapparency inapparent infection	Inapparenz inapparente Infektion	2

het ontbreken van zichtbare symptomen, de infectie wordt niet apparent of zichtbaar, manifesteert zich niet.

internodiënverkorting	internode shortening shortening of internodes	Internodienverkürzung	28

verkorting van stengelleden; leidt tot dubbelknopigheid (zie aldaar) of rozetvorming (zie aldaar).

kakelbont	calico	Kaliko	1

schrille, zeer opvallende, met aucubamozaïek overeenkomende kleurschakering, waarbij echter de brilliant geel tot wit gekleurde delen van het blad overheersen.

kanker	canker	Krebs	25

tumorachtige, met necrose gepaard gaande weefselwoekering op stammen en takken van houtachtige gewassen, waarbij het dode bastgedeelte door een callusachtige woekering is omgeven (een in de literatuur niet scherp omschreven verschijnsel, dat bij virusziekten nog onvoldoende is onderzocht).

kiemrustreductie	reduction of seed dormancy	Keimruhereduktion	32

het voortijdig uitlopen van zaden of pootgoed, soms reeds in onrijpe toestand nog aan de moederplant.

krenterigheid (pseudo netnecrose)	tuber blotching (pseudo net necrosis)	Pseudonetznekrose	18

roest- tot donkerbruine vlekken en stippen in het vruchtvlees van aardappelknollen (veroorzaakt door aucuba virus).

kringerigheid	corky ring spot cf. ring spot	Pfropfenbildung Kringerigkeit	18

bij aardappelknollen op doorsnede zichtbare kring- en boogvormige, door necrose en kurkvorming zwartbruin gekleurde figuren (veroorzaakt door stengelbontvirus).

kringvlek	ring spot	Ringfleck	11, 18

vlek, waarin concentrische kringen van licht en donker gekleurd weefsel elkaar afwisselen; vaak zijn de donkere kringen necrotisch en licht gekleurde delen vergeeld of verdroogd. Ze kunnen ook enkelvoudig zijn.

kurkvorming	cork formation	Korkbildung	7,2

laagsgewijze celvermeerdering, waarbij de wanden der nieuwgevormde cellen verkurken; een overigens normaal verschijnsel, dat ingeval van ziekte in abnormale mate en op abnormale plaatsen vaak als wondreactie optreedt.

latentie	latency	Latenz	2

die vorm van inapparentie, waarbij de infectie blijvend onzichtbaar is (een dergelijke waardplant wordt carrier of smetstofdrager genoemd).

lokale lesie	local lesion	Lokalläsion Lokalherd Einzelherd	4

verkleurde, verdroogde of necrotische vlek van doorgaans beperkte omvang, ontstaan op de plaats van infectie.

lokale symptomen primaire symptomen	local symptoms primary symptoms	Lokalsymptomen Primärsymptomen Initialsymptomen	4

op de plaats van infectie ontstaande symptomen.

maskering	masking	Maskierung	2

het tijdelijk afwezig zijn van symptomen, bv. tengevolge van uitwendige omstandigheden; tijdelijke inapparentie.

misvorming	malformation deformation	Missbildung Deformation	21 e.v

afwijking in vorm en bouw van delen van de plant of van de gehele plant. Histoïde misvormingen bestaan uit abnormaal gebouwde weefsels of organen. Organoïde misvormingen zijn gekenmerkt door een afwijkende uitwendige bouw, dit zijn de morfologische afwijkingen of teratomata (zie aldaar).

mozaïek	mosaic	Mosaik	10

kleurschakering, waarbij de lichtgroene, vergeelde of soms bijna witte delen onregelmatig over het blad zijn verdeeld, terwijl de lichte en donker gekleurde delen meestal scherp begrensd en hoekig van vorm zijn. Mozaïek is vaak niet scherp te scheiden van vlekking, gevlektheid of bontheid (zie aldaar).

naaldbladigheid zie draadbladigheid			26

necrose	necrosis	Nekrose	7,1 e.v

het afsterven van cellen, celgroepen, weefsel of plantedelen, waarbij steeds een scherpe grens bestaat tussen dode en levende delen.

negatieve geotropie, versterking van de -	negative geotropy, intensification of -	negative Geotropie, Verstärkung der -	32

neiging van organen, voornamelijk stengelorganen, om een steile, opgerichte stand in te nemen.

nerfbandmozaïek	vein banding	Adernbänderung Adernbandmosaik	11

nerfmozaïek, waarbij het vergeelde of soms het donker gekleurde weefsel in stroken of banden langs de grotere nerven voorkomt.

nerfglazigheid	vein clearing	Adernaufhellung	10

het doorzichtig of glazig worden van de nervatuur, waardoor deze lichter dan normaal gekleurd tegen de rest van het blad afsteekt.

nerfmozaïek	vein mosaic	Adernmosaik	11

vergeling van onregelmatig gerangschikte delen van, tot soms de gehele nervatuur, met inbegrip van onregematig begrensde delen van het aangrenzend bladweefsel.

nerfvergeling (geelnervigheid) — vein yellowing — Adernvergilbung — 9

regelmatige vergeling van de bladnervatuur met soms iets van het aangrenzende bladmoes; het patroon is echter zeer regelmatig.

netnecrose — net necrosis — Netznekrose — 18

afsterving van zeefvaten en begeleidende cellen in de vaatbundels van aardappelknollen, zichtbaar op doorsnede van de knol als zwarte, onregelmatig verdeelde, netvormige tekening (veroorzaakt door bladrolvirus).

olievlek — oil fleck — Ölfleck — 11

licht gekleurde bladvlek, die geheel bestaat uit, of omgeven is door een rand van enigszins doorzichtig, glazig bladmoes.

onvolledige verhouting — defective lignification — unvollständige Verholzung — 20

verdikking van de houtelementen (xyleem) met celstof (cellulose) in plaats van met houtstof (lignine), waardoor houtige takken (van appel) een ongewone buigzaamheid vertonen (zie rubberhout symptomen).

oogstreductie
zie opbrengstdaling — 8

opbrengstdaling — yield reduction — Ertragsabnahme — 8
oogstreductie

vermindering van de opbrengst.

organoïde misvorming
zie misvorming — 21

overgevoeligheid — hypersensitivity — Uberempfindlichkeit — 15

necrotische reactie van de geïnfecteerde en eventueel naburige cellen, waardoor de infectie blijft gelocaliseerd en een systemische reactie uitblijft.

primaire symptomen
zie lokale symptomen — 4

prolificatie (doorgroei) — prolification — Prolifikation (Durchwachsung) — 29,

de vorming van een bebladerd takje of soms een bloemgestel of bloem in het centrum van de bloem, dus op de top van de bloembodem (centrale prolificatie) en/of in de oksels van bloemorganen (laterale prolificatie).

pseudo netnecrose
zie krenterigheid — 18

randvergeling — edge yellowing — Randvergilbung — 9
bladrandvergeling — Blattrandvergilbung

regelmatige vergeling van de randgedeelten van het blad, min of meer tot de rand beperkte chlorose van het blad.

rozetvorming — rosetting — Rosettenbildung — 28

opeenhoping van bladeren aan stengeltop(pen) ten gevolge van internodiënverkorting (zie aldaar).

rubberhout — rubbery wood — Gummiholz — 20

ongewone buigzaamheid van houtige takken ten gevolge van onvolledige verhouting (zie aldaar) van het xyleem.

ruwschilligheid — rough skin — Rauhschaligkeit — 21

ruwheid van de schil (bv. van appels) ten gevolge van oppervlakkige (soms versterkte) kurkvorming (vergelijk schubbigheid).

schubbigheid — bark scaling — Schuppigkeit — 21

het in schubben of schilfers afstoten van de bast ten gevolge van inwendige kurkvorming.

secondaire symptomen — 4
zie systemische symptomen

smalbladigheid — leaf narrowing — Schmalblättrigkeit — 26

versmalling van de bladschijf.

spruitzwelling — shoot swelling — Sprossschwellung — 25

opzwelling van spruiten of takken of van gedeelten hiervan.

steriliteit — sterility — Sterilität — 32

het achterwege blijven van de vorming van vruchten en/of zaden en van stuifmeel; meestal is het een gevolg van het min of meer vegetatief worden van vruchtbladen en meeldraden bij bloemoplossing (zie fyllodie).

symptoom, ziekte - — symptom, disease - — Symptom, Krankheits - — 1

in de plant optredende waarneembare afwijking, ontstaan als gevolg van de interactie waardplant--ziekteverwekker. De gezamenlijke symptomen of ziekteverschijnselen vormen het syndroom of ziektebeeld (zie aldaar).

syndroom
ziektebeeld — syndrome — Krankheitsbild — 1

het geheel van de door een ziekteverwekker in een bepaalde plant veroorzaakte symptomen (zie aldaar).

systemische symptomen secondaire symptomen	systemic symptoms secondary symptoms	systemische Symptomen Folgesymptomen Sekondärsymptomen	4

verschijnselen, welke enige tijd na infectie in de niet rechtstreeks geïnoculeerde delen van de plant ontstaan.

takafplatting	flattening of branches	Flachästigkeit	25

onregelmatige afplatting van de twee à drie tot meer jaren oude takken (bij appel).

teratomata	teratomata	Teratomata	26

een soms gebruikte benaming voor morfologische of organoïde afwijkingen (zie misvormingen), daar deze worden bestudeerd in de teratologie.

thyllenvorming, overmatige -	tyloses, excessive formation of -	Thyllenbildung, übermässige -	19

de overmatige en vroegtijdige vorming van thyllen in het nog levende hout, waardoor stagnatie in het watertransport optreedt.

topnecrose	top necrosis	Spitzennekrose	16

het afsterven van de top der plant.

tumor	tumor	Tumor	23

gezwel of histoïde misvorming of opzwelling, ontstaan ten gevolge van een plaatselijke, vaak zeer onregelmatige woekering van cellen en/of weefsels.

tussennervig mozaïek	interveinal mosaic	zwischennerviges Mosaik	11

mozaïek, waarbij de licht gekleurde gedeelten hoofdzakelijk tussen de nerven voorkomen.

uitwas zie enatie			24,2

verdorring	withering	Verdorrung	14

het geheel verdrogen van plantedelen, waardoor bv. het blad verschrompelt.

vergeling	yellowing	Vergilbung	9

het gedeeltelijk of geheel verdwijnen van de groene kleur ten gevolge van chlorofylafwijkingen, waardoor de gele kleur der xanthofyllen en carotenen tot uiting komt; de gele kleur is soms abnormaal versterkt door een toename in gehalte aan xanthofyllen en carotenen (zie chlorose, mozaïek en bonthaid).

vergroening — greening — Vergrünung — 13,3
virescentie — virescence — Vireszenz

het groen worden van bloemdelen, welke in normale toestand wit of gekleurd zijn, het is doorgaans het eerste stadium van bloemoplossing (zie aldaar).

verwelking — wilting — Welke — 14

het slap worden van plantedelen door verlies van turgor.

virescentie — 13,3
zie vergroening

vlekking — mottling — Scheckung — 11
gevlektheid — Buntblättrigkeit
bontheid

kleurschakering waarbij de kleurafwijking beperkt is tot verspreide, min of meer ronde vlekken, stippen of spikkels, die niet altijd scherp begrensd zijn. Vlekking is niet altijd scherp te scheiden van mozaïek (zie aldaar).

waaierbladvorming — fan leaf formation — Fächerblattbildung — 26

een vorm van smalbladigheid in handnervige bladeren, waarbij de grote nerven als in een gedeeltelijk gesloten waaier dicht opeen komen te staan.

woekering — proliferation — Wuckerung — 29

ongecorreleerde celvermeerdering of weefselontwikkeling bv. bij tumorvorming.

zetmeelophoping — accumulation of starch — Stärkespeicherung — 19

abnormale ophoping van zetmeel in bladeren ten gevolge van een verstoorde afvoer; de bladeren zijn daardoor bij aanvoelen stugger en dikker dan normaal.

ziektebeeld — 1
zie syndroom

zigzag-groei — zig zag growth — Zickzackwuchs — 28

zigzagvormige stengelgroei.

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