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THE FILTERABLE VIRUSES

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SUPPLEMENT NO. 2

ORDER VIRALES
THE FILTERABLE VIRUSES

FILTERABLE VIRUSES*

The so-called filterable viruses, today generally called merely viruses, are still of unknown affiliations so far as relationships to established groups of microorganisms are concerned. They are treated here as members of an order, consisting of 13 families, 32 genera and 248 species.

Among viruses as we know them, there are three constituent groups that have come to be recognized, and to some extent named and classified, through the largely separate efforts of bacteriologists, animal pathologists, and plant pathologists. Taxonomic overlapping of the three groups, viruses affecting bacteria, viruses having only animal hosts, and viruses invading higher plants, can hardly be justified as yet by available evidence. Nevertheless it has been shown that a single virus may multiply both in a plant host and in an insect vector. This seems to dispose of the thought that adaptation to a plant or animal environment would necessarily preclude utilization of other sources of the materials needed for multiplication.

For the present it seems feasible to continue with the custom, tacitly accepted in the past, of classifying bacteriophages separately as one sub-group, viruses causing diseases in seed plants as a second sub-group, and those causing diseases in animals as a third sub-group. It should be recognized that this may prove to be only a temporary arrangement, necessary because we have no evidence to warrant taxonomic overlapping of the three groups and useful while we await critical investigations and possible development of a substitute plan capable of displaying natural relationships to better advantage. Eventually evidence may become available to show that some bacteriophages can infect higher plants or animals and can increase in the new environment, or that viruses known to attack animals or plants can similarly enlarge their host ranges. Or, there may be discoveries of common physical properties that would aid in formulating an interlocking classification, for which at present we lack any substantial basis.

It is of especial significance now that the three fields be unified at least by a parallel development of nomenclature. Toward this end the present section of this supplement is directed.

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* Supplement No. 2 has been prepared by Francis O. Holmes, The Rockefeller Institute for Medical Research, Princeton, N. J., September, 1944. In this section, authorities for the names of plant hosts are in general as given by Gray's New Manual of Botany, 7th edition, and Bailey's Manual of Cultivated Plants, 1938; in each of these standard works will be found a list of abbreviations customarily used in botany in citing authorities for binomials.

ORDER VIRALES Breed, Murray and Hitchens.

(Jour. Bact., *47*, 1944, 421.)

Viruses. Etiological agents of disease, typically of small size and capable of passing filters that retain bacteria, increasing only in the presence of living cells, giving rise to new strains by mutation, not arising *de novo*. A considerable number of viruses have not been proved filterable; it is nevertheless customary to include these viruses with those known to be filterable, because of similarities in other attributes and in the diseases induced. Some not known to be filterable are inoculable only by special techniques, as by grafting or by use of insect vectors, and suitable methods for testing their filterability have not been developed; moreover, it is not certain that so simple a criterion as size measured in terms of filterability will prove to be an adequate indicator of the limits of the natural group. Cause diseases of bacteria, plants and animals.

Key to the suborders of order **Virales.**

I. Infecting bacteria.
Suborder I. *Phagineae*, p. 1128.
II. Infecting higher plants.
Suborder II. *Phytophagineae* p. 1145.
III. Infecting animals (insects, mammals).
Suborder III. *Zoophagineae*, p. 1225.

SUBORDER I. Phagineae *subordo novus*.

Viruses pathogenic in bacteria; bacteriophages. Containing at present only one family, the *Phagaceae*.

FAMILY I. PHAGACEAE HOLMES.

(Handb. Phytopath. Viruses,* 1939, 1.)

Characters those of the suborder. There is a single genus.

Genus I. Phagus *Holmes.*

(*Loc. cit.*, 1.)

Characters those of the family. Generic name from Greek *phagein*, to eat.

The type species is *Phagus minimus* Holmes.

NOTE: *Bacteriophagum* d'Herelle (Compt. rend. Soc. Biol., Paris, *81*, 1918, 1161) a genus name applied in connection with early studies of bacteriophages, had as its type species *Bacteriophagum intestinale* d'Herelle, a bacteriophage that is not now identifiable or, more probably, a mixture of such unidentifiable bacteriophages, for filtrates containing it were said to be capable of killing outright a culture of bacteria (*ibid.*, 1160). The genus name *Bacteriophagum* is, therefore, regarded as a *nomen dubium*, if not also a *nomen confusum;* subsequently it was abandoned by its author, for reasons that are not clear, in favor of the genus name *Protobios* d'Herelle 1924 (Immunity in natural infectious disease; page 343 of authorized English edition by George H. Smith, Baltimore, Williams & Wilkins Co., 1924, 399 pp). *Protobios protobios*

* Holmes, F. O., Handbook of Phytopathogenic Viruses, Burgess Publishing Company, Minneapolis, Minn., 1939, 221 pp.

d'Herelle (*loc. cit.*, 345), presumably the type species of this genus, was not an ordinary virus but was said to be non-parasitic (i.e., free-living) in nature, was capable of reducing sulphur, and is not now identifiable. The genus name *Protobios* and the corresponding binomial *Protobios bacteriophagus* d'Herelle are therefore regarded also as *nomina dubia* and are not used here. *Bacteriophagus* Thornberry (Phytopath., *31*, 1941, 23) appears to represent a variant spelling of d'Herelle's earlier genus name; it was not accompanied by any indication of what recognizable single bacteriophage served as type and thus does not modify the standing of *Bacteriophagum*.

Key to the species of genus **Phagus.**

I. Dysentery-coli bacteriophages.

A. Producing large plaques, 8 to 12 mm in diameter.

1. Particle size small, 8 to 12 millimicrons.

1. *Phagus minimus.*

2. Particle size 15 to 20 millimicrons.

2. *Phagus minor.*

B. Producing moderately large plaques, 2 to 6 mm in diameter, with distinct halo.

1. Particle size 20 to 30 millimicrons.

3. *Phagus parvus.*
4. *Phagus primarius.*
5. *Phagus secundarius.*
6. *Phagus dysenteriae.*

C. Plaques medium size, 1 to 3 mm in diameter, with distinct halo.

1. Particle size 25 to 40 millimicrons.

7. *Phagus medius.*
8. *Phagus astrictus.*

D. Plaques small, 0.5 to 1.5 mm in diameter, with soft edge or narrow halo.

1. Particle size 30 to 45 millimicrons.

9. *Phagus major.*
10. *Phagus coli.*
11. *Phagus artus.*

E. Plaques very small, 0.1 to 1.2 mm in diameter, with sharp edges.

1. Particle size 50 to 75 millimicrons.

12. *Phagus maximus.*

II. Bacteriophages attacking *Agrobacterium tumefaciens* Conn, *Pseudomonas solanacearum* Smith, *Xanthomonas citri* Dowson, *Xanthomonas pruni* Dowson, *Erwinia carotovora* Holland, *Erwinia aroideae* Holland, *Bacterium stewarti* E. F. Smith.

A. Specific for bacterial hosts named above.

13. *Phagus tumoris.*
14. *Phagus solanacearum.*
15. *Phagus citri.*
16. *Phagus pruni.*
17. *Phagus deformans.*
18. *Phagus contumax.*
19. *Phagus maidis.*

III. Bacteriophages attacking *Salmonella enteritidis* Castellani and Chalmers.

20. *Phagus enteritidis.*
21. *Phagus commutabilis.*
22. *Phagus tertius.*
23. *Phagus dubius.*

IV. Bacteriophage attacking *Salmonella typhosa.*

24. *Phagus indicens.*

V. Bacteriophages attacking *Bacillus megatherium* DeBary, *Bacillus mycoides* Flügge, and *Rhizobium leguminosarum* Frank.

A. Thermal inactivation at 75° C in 10 minutes *in vitro.*

1. Host may be freed from bacteriophage by heating at 80° C for 10 minutes.

25. *Phagus testabilis.*

2. Host retains virus even when heated at 90° C for 10 minutes.

26. *Phagus indomitus.*

B. Thermal inactivation at 60° C in 30 minutes.

27. *Phagus subvertens.*

VI. Bacteriophages attacking streptococci.

28. *Phagus ineptus.*
29. *Phagus streptococci.*
30. *Phagus maculans.*
31. *Phagus lacerans.*
32. *Phagus tolerans.*
33. *Phagus michiganensis.*

VII. Bacteriophages attacking staphylococci.

34. *Phagus fragilis.*
35. *Phagus intermedius.*
36. *Phagus caducus.*
37. *Phagus alpha.*
38. *Phagus beta.*

39. *Phagus durabilis.*
40. *Phagus liber.*

VIII. Bacteriophages attacking vibrios.

41. *Phagus cholerae.*
42. *Phagus celer.*
43. *Phagus effrenus.*
44. *Phagus lentus.*

IX. Bacteriophages attacking *Corynebacterium diphtheriae* Lehmann and Neumann.

45. *Phagus diphtheriae.*
46. *Phagus futilis.*

1. **Phagus minimus** Holmes. (Handb. Phytopath. Viruses, 1939, 141.) From Latin *minimus*, least, in reference to size.

Common name: Bacteriophage S13.

Hosts: *Escherichia coli* Castellani and Chalmers; *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: On plate cultures that are uniformly covered with confluent colonies of host organisms, this bacteriophage produces large cleared plaques, 8 to 12 mm in diameter, with wide shelving edges.

Serological relationships: No cross-neutralization reactions with bacteriophages C13, C36, D5, D20, C18, D3, S8, C21, C16, and D6.

Immunological relationships: Member of Resistance Group I.

Other properties: Particle size 8 to 12 millimicrons. Not affected by 26.3 per cent urea solution. Little or no inactivation by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candle-power light for 30 minutes. Lysis completely inhibited by 0.25 per cent solution of sodium citrate.

Literature: Burnet and McKie, Jour. Path. and Bact., *36*, 1933, 299–306, 307–318; *37*, 1933, 179–184; Burnet et al., Austral. Jour. Exp. Biol. and Med. Sci., *15*, 1937, 227–368.

2. **Phagus minor** H. (*loc. cit.*, 141). From Latin *minor*, lesser.

Common names: Bacteriophage C13, C8, and D44.

Hosts: *Escherichia coli* Castellani and Chalmers; *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Large plaques, 8 to 12 mm in diameter, with wide shelving edges.

Serological relationships: Cross reactions with bacteriophages C8 and D44 but not with bacteriophages S13, C36, D5, D20, D13, C18, D3, S8, C21, C16, D6.

Immunological relationships: Member of Resistance Group I.

Other properties: Particle size, 15 to 20 millimicrons. Completely inactivated by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candle-power light for 30 minutes. Specific soluble substance formed in lysed cultures blocks phage-antiphage reaction.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318; Brit. Jour. Exp. Path., *14*, 1933, 100–108.

3. **Phagus parvus** H. (*loc. cit.*, 142). From Latin *parvus*, small.

Common names: Bacteriophage C36, S18, C38, M, and C37 of Burnet.

Hosts: *Escherichia coli* Castellani and Chalmers; *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Moderately large plaques, 2 to 6 mm in diameter, with distinct halo.

Serological relationships: Induces formation of antibody capable of neutralizing bacteriophages S18, C38, M, and C37, but not bacteriophages S13, C13, D5, D20, D13, C18, D3, S8, C21, C16, or D6, which represent distinct serological groups.

Immunological relationships: Member of Resistance Group I.

Other properties: Particle size, 20 to 30 millimicrons. Completely inactivated by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candle-power light for 30 minutes.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318.

4. **Phagus primarius** H. (*loc. cit.*, 143). From Latin *primarius*, chief or first.

Common names: Bacteriophage D5, C51, C50, and D48.

Hosts: *Escherichia coli* Castellani and Chalmers; *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Moderately large plaques, 2 to 6 mm in diameter, with distinct halos.

Serological relationships: Cross-neutralization reactions with bacteriophages C51, C50, and D48, but not with S13, C13, C36, D20, D13, C18, D3, S8, C21, C16, D6.

Immunological relationships: Member of Resistance Group I.

Other properties: Particle size, 20 to 30 millimicrons. Completely inactivated by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candle-power light for 30 minutes.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318.

5. **Phagus secundarius** H. (*loc. cit.*, 143). From Latin *secundarius*, inferior or second.

Common names: Bacteriophage D20 and G.

Hosts: *Escherichia coli* Castellani and Chalmers; *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Moderately large plaques, 2 to 6 mm in diameter, with distinct halo.

Serological relationships: No cross-neutralization reactions with bacteriophages S13, C13, C36, D5, D13, C18, D3, S8, C21, C16, or D6.

Immunological relationships: Member of Resistance Group II.

Other properties: Nearly all inactivated by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candle-power light for 30 minutes. Particle size, 20 to 30 millimicrons.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318.

6. **Phagus dysenteriae** H. (*loc. cit.*, 144). From Latin *dysenteria*, dysentery.

Common names: Bacteriophage D13, specific dysentery phage.

Host: *Shigella dysenteriae* Castellani and Chalmers.

Insusceptible species: *Escherichia coli* Castellani and Chalmers.

Induced disease: Moderately large plaques, 2 to 6 mm in diameter, with distinct halo.

Serological relationships: Antiserum to this strain is not known to be effective against any other strain of bacteriophage; in particular, no cross reactions with bacteriophages S13, C13, C36, D5, D20, C18, D3, S8, C21, C16, or D6.

Immunological relationships: Member of Specific Dysentery Resistance Group.

Other properties: Particle size, 20 to 30 millimicrons. Completely inactivated by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candle-power light for 30 minutes.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318.

7. **Phagus medius** H. (*loc. cit.*, 144). From Latin *medius*, moderate, in reference to particle size.

Common name: Bacteriophage C18, C35, C26, C47, or C34.

Hosts: *Escherichia coli* Castellani and Chalmers; *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Medium size plaques, 1 to 3 mm in diameter, with distinct halo.

Serological relationships: Cross reactions with bacteriophages C35, C26, C47, and C34, but not with S13, C13, C36, D5, D20, D13, D3, S8, C21, C16, or D6.

Immunological relationships: Member of Resistance Group II.

Other properties: Particle size, 25 to 40 millimicrons.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318.

8. **Phagus astrictus** H. (*loc. cit.*, 145). From Latin *astrictus*, limited, in reference to inability to lyse *Escherichia coli* Castellani and Chalmers.

Common names: Bacteriophage D3; "smooth" dysentery phage.

Host: *Shigella dysenteriae* Castellani and Chalmers.

Insusceptible species: *Escherichia coli* Castellani and Chalmers.

Induced disease: Medium size plaques, 1 to 3 mm in diameter, with distinct halo.

Serological relationships: No cross-neutralization reactions with bacteriophages S13, C13, C36, D5, D20, D13, C18, S8, C21, C16, or D6.

Immunological relationships: Member of Smooth Dysentery Resistance Group.

Other properties: Particle size, 25 to 40 millimicrons. Nearly all inactivated by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candle-power light for 30 minutes.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318.

9. **Phagus major** H. (*loc. cit.*, 146). From Latin *major*, greater, in reference to particle size.

Common name: Bacteriophage S8, L, S28, C33, or S41.

Hosts: *Escherichia coli* Castellani and Chalmers; *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Small plaques, 0.5 to 1.5 mm in diameter, with soft edge or narrow halo.

Serological relationships: No cross-neutralization reactions with bacteriophages S13, C13, C36, D5, D20, D13, C18, D3, C21, C16, or D6.

Immunological relationships: Member of Resistance Group I.

Other properties: Particle size, 30 to 45 millimicrons.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318; Brit. Jour. Exp. Path., *14*, 1933, 100–108; Gough and Burnet, *ibid.*, *38*, 1934, 301–311.

10. **Phagus coli** H. (*loc. cit.*, 146). From Latin *colon*, the colon.

Common names: Bacteriophage C21 or C5; specific *coli* phage.

Host: *Escherichia coli* Castellani and Chalmers.

Insusceptible species: *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Small plaques, 0.5 to 1.5 mm in diameter, with soft edge or very narrow halo.

Serological relationships: No cross-neutralization with bacteriophages S13, C13, C36, D5, D20, D13, C18, D3, S8, C16, or D6.

Immunological relationships: Member of Specific *Escherichia coli* Resistance Group.

Other properties: Particle size, 30 to 45 millimicrons. Completely inactivated by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candle-power light for 30 minutes.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318.

11. **Phagus artus** H. (*loc. cit.*, 148). From Latin *artus*, narrow, in reference to limited host range.

Common names: Bacteriophage D6, D33; smooth dysentery phage.

Host: *Shigella dysenteriae* Castellani and Chalmers, smooth strains.

Induced disease: Small plaques, 0.5 to 1.5 mm in diameter, with soft edge or very narrow halo.

Serological relationships: Not neu-

tralized by sera specific for bacteriophages S13, C13, C36, D5, D20, D13, C18, D3, S8, C21, or C16.

Immunological relationships: Member of Smooth Dysentery Resistance Group.

Other properties: Particle size, 30 to 45 millimicrons.

Literature: Burnet, Jour. Path. and Bact., *36*, 1933, 307–318.

12. **Phagus maximus** H. (*loc. cit.*, 147). From Latin *maximus*, greatest, in reference to particle size.

Common names: Bacteriophage C16, C4, C15, C20, C32, C46, D4, D12, D29, D53, H, J, K, and W. L. L.

Hosts: *Escherichia coli* Castellani and Chalmers; *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Small plaques, 0.1 to 1.2 mm in diameter, with sharp edges.

Serological relationships: No cross-neutralization reaction with bacteriophages S13, C13, C36, D5, D20, D13, C18, D3, S8, C21, D6, or staphylococcus bacteriophage Au2. Agglutinated and inactivated by homologous, though not by other, antisera. For agglutination an original titer of 2×10^9 or higher is required; the reaction is visible to the unaided eye after 24 hours at 50° C and succeeds even after inactivation by heat (70 to 85° C for 30 minutes), formaldehyde, or a photodynamic dye (proflavine).

Immunological relationships: Member of Resistance Group II.

Thermal inactivation: At or below 70° to 85° C for 30 minutes.

Other properties: Particle size estimated by filtration as 50 to 75 millimicrons, by centrifuging as 79 to 90 millimicrons, from photographs as 50 to 60 millimicrons. Rapidly inactivated by 26.3 per cent urea solution. Little or no inactivation by 1:25,000 methylene blue in 2 mm layer 20 cm from 100 candlepower light for 30 minutes. Lysis not inhibited by 1.5 per cent or weaker solutions of sodium citrate. Thermolabile specific soluble substance formed in lysed cultures blocks phage-antiphage reaction.

Literature: Burnet, Brit. Jour. Exp. Path., *14*, 1933, 93–100, 100–108, 302–308; Jour. Path. and Bact., *36*, 1933, 307–318; *37*, 1933, 179–184; Burnet and Lush, *ibid.*, *40*, 1935, 455–469; Burnet and McKie, *ibid.*, *36*, 1933, 299–306.

13. **Phagus tumoris** H. (*loc. cit.*, 150). From Latin *tumor*, a swelling, in reference to association of this bacteriophage with bacterial tumors.

Common name: *Agrobacterium tumefaciens* bacteriophage.

Host: *Agrobacterium tumefaciens* Conn, most strains.

Insusceptible species: Some strains of *Agrobacterium tumefaciens*, *Bacterium stewarti* E. F. Smith, *Erwinia atroseptica* Bergey et al., *E. carotovora* Holland, *Pseudomonas tabaci* Stapp, *Xanthomonas beticola* Burkholder, *X. campestris* Dowson, *X. citri* Dowson, *X. phaseoli* Dowson *X. pruni* Dowson and *X. vesicatoria* Dowson.

Geographical distribution: United States, Russia.

Induced disease: Plaques 2 to 6 mm in diameter in 4 to 6 hours, edges of plaques spotted, moth-eaten in appearance until 40 hours after seeding; enlargement then stops and the edges of the plaques become smooth, double-ringed. Infection of plants by *Agrobacterium tumefaciens* is progressively inhibited by increasing amounts of bacteriophage in inoculum.

Thermal inactivation: At 95° C in 10 minutes (another report says 70° C, time not recorded).

Other properties: Resists dilution to $1:10^{11}$; storage at 5° C for over 25 months; prompt, though not gradual, drying; 1 per cent hydrogen peroxide for 72 hours; 95 per cent ethyl alcohol for 1 hour; 70 per cent ethyl alcohol for 6 hours; 2½ per cent phenol for 1 hour; 1:3000 nitric acid for 1 hour; N/64 sodium hydroxide for 1 hour.

Literature: Israilsky, Cent. f. Bakt.,

II Abt., *67*, 1926, 236–242; *71*, 1927, 302–311; *79*, 1929, 354–370; Kent, Phytopath., *27*, 1937, 871–902; Muncie and Patel, Phytopath., *20*, 1930, 289–305.

14. **Phagus solanacearum** H. (*loc. cit.*, 148). From name of host.

Common name: *Pseudomonas solanacearum* bacteriophage.

Host: *Pseudomonas solanacearum* Smith.

Geographical distribution: Formosa (Taiwan).

Induced disease: Medium size plaques on plate cultures of *Pseudomonas solanacearum*.

Serological relationships: When injected into rabbits, this bacteriophage stimulates the production of a specific precipitating antibody not giving cross reactions with anti-bacterial antibodies. Antiphagic serum inactivated at 90° C in 10 minutes.

Thermal inactivation: At 63° C in 10 minutes (61° C in 30 minutes; 66° C in about 1 minute).

Other properties: Optimum temperature for increase, 34° C.

Literature: Matsumoto and Okabe, Jour. Plant Prot., *22*, 1935, 15–20; Jour. Soc. Trop. Agr., 7, 1935, 130–139; *9*, 1937, 205–213.

15. **Phagus citri** H. (*loc. cit.*, 149). From name of host.

Common name: *Xanthomonas citri* bacteriophage.

Host: *Xanthomonas citri* Dowson, the citrus canker organism.

Geographical distribution: Formosa (Taiwan).

Induced disease: Lysis. This bacteriophage has been isolated from soil under diseased trees, and once from infected leaves. It may play a role in the destruction of the citrus canker organism in the soil.

Other properties: Optimum temperature for increase, 30° C.

Literature: Matsumoto and Okabe, Agriculture and Horticulture, *12*, 1937, 2055–2059.

16. **Phagus pruni** H. (*loc. cit.*, 151). From name of host.

Common name: *Xanthomonas pruni* bacteriophage.

Host: *Xanthomonas pruni* Dowson.

Geographical distribution: United States (from soil beneath infected peach trees).

Induced disease: Lysis in broth cultures; plaques on agar cultures, but characteristics of plaques not described.

Other properties: Estimated diameter 11 millimicrons in broth. Resists dilution to $1:10^6$ or more.

Literature: Anderson, Phytopath., *18*, 1928, 144; Thornberry, *ibid.*, *25*, 1935, 938–946.

17. **Phagus deformans** H. (*loc. cit.*, 151). From Latin *deformare*, to disfigure, in reference to malformation of infected host cells.

Common name: *Erwinia carotovora* bacteriophage.

Host: *Erwinia carotovora* Holland.

Insusceptible species: *Agrobacterium tumefaciens* Conn, except in some early tests with possibly mixed bacteriophages; *Erwinia amylovora* Winslow et al., *E. melonis* Holland, *Salmonella pullorum* Bergery et al., *S. gallinarum* Bergey et al., Shigella dysenteriae Castellani and Chalmers, *Xanthomonas pruni* Dowson.

Geographical distribution: United States (Michigan).

Induced disease: In *Erwinia carotovora*, cells reduced in motility, agglutinated, malformed, some elongated, others swollen, bulged at one end, bulged in middle, or enlarged and spherical.

Other properties: Resists dilution to $1:10^9$, and storage in sterile medium at room temperature for 5½ months.

Literature: Coons and Kotila, Phytopath., *15*, 1925, 357–370; Mallmann and Hemstreet, Jour. Agr. Res., *28*, 1924, 599–602.

18. **Phagus contumax** *spec. nov.* From Latin *contumax*, refractory, in reference to ability of this bacteriophage to withstand heating sufficient to destroy accompanying host cells.

Common name: *Erwinia aroideae* bacteriophage.

Host: *Erwinia aroideae* Holland.

Insusceptible species: *Agrobacterium tumefaciens* Conn, *Bacterium formosanum* Okabe, *Erwinia carotovora* Holland, *Pseudomonas andropogoni* Stapp, *P. solanacearum* Smith, *P. tomato* Burkholder, *Xanthomonas campestris* Dowson, *X. citri* Dowson, *X. malvacearum* Dowson, *X. nakatae* Dowson, *X. phaseoli* Dowson, *X. ricinicola* Dowson.

Geographical distribution: Formosa (Taiwan).

Induced disease: Very small plaques, 0.1 to 1.0 mm (mostly less than 0.5 mm) in diameter.

Thermal inactivation: Resists heating at 60° C for 30 minutes without appreciable loss of titer, but host organism is killed by this treatment.

Other properties: Optimum temperature for increase, about 25° C. This bacteriophage may be prepared by heating centrifuged cultures at 60° C for 30 minutes as efficiently as by filtration to remove bacteria.

Literature: Matsumoto, Trans. Nat. Hist. Soc. Formosa, *29*, 1939, 317–338; *30*, 1940, 89–98; *31*, 1941, 145–154; Matsumoto and Sawada, *ibid.*, *28*, 1938, 247–256.

19. **Phagus maidis** H. (*loc. cit.*, 152). From New Latin *mais*, corn (maize), host of *Bacterium stewarti*.

Common name: *Bacterium stewarti* bacteriophage; *Phytomonas stewarti* bacteriophage; *Aplanobacter stewarti* bacteriophage.

Host: *Bacterium stewarti* E. F. Smith (= *Pseudomonas stewarti* E. F. Smith, *Phytomonas stewarti* Bergey et al. and *Aplanobacter stewarti* McCulloch).

Geographical distribution: United States

Induced disease: In *Bacterium stewarti*, variation or loss of yellow color, change of viscosity of growth, reduction or loss of virulence. Infection of corn plants by seed-borne *Bacterium stewarti* is much reduced by treating seeds with this bacteriophage before they are planted.

Thermal inactivation: Above 65° C in 30 minutes.

Other properties: Infective in dilutions to 10^{-7}. Soon lost from cultures maintained at pH 3.85 to 4.00, or on Ivanoff's medium, which contains oxidizing compounds.

Literature: Thomas, Phytopath., *25*, 1935, 371–372; Science, *88*, 1938, 56–57; Phytopath., *30*, 1940, 602–611.

20. **Phagus enteritidis** H. (*loc. cit.*, 153). From name of host.

Common names: *Salmonella enteritidis* bacteriophage 1, 12, or 33; Group A bacteriophages.

Hosts: *Salmonella enteritidis* Castellani and Chalmers, *S. gallinarum* Bergey et al., *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Plaques of medium size, usually with surrounding translucent halo.

Immunological relationships: Member of Resistance Group A; host individuals that have acquired resistance to this bacteriophage are resistant to lines 12 and 33, but susceptible to *Salmonella enteritidis* bacteriophages 8, 20, and 11, as well as to other strains of Resistance Groups B, C, and D.

Literature: Burnet, Jour. Path. and Bact., *32*, 1929, 15–42.

21. **Phagus commutabilis** H. (*loc. cit.*, 153). From Latin *commutabilis*, variable, in reference to differences within Resistance Group B, typified by this bacteriophage.

Common names: *Salmonella enteritidis* bacteriophage 8, 18, 28, 31, 34, 38; Group B bacteriophages.

Hosts: *Salmonella enteritidis* Castellani

and Chalmers, *Shigella dysenteriae* Castellani and Chalmers, *Shigella gallinarum* Weldin, *Salmonella typhosa* White.

Induced disease: Small plaques with sharp edges, or moderately large plaques with characteristic halo.

Immunological relationships: Member of Resistance Group B; host individuals that have acquired resistance to this bacteriophage are resistant to lines 18, 28, 31, 34, and 38, but susceptible to *Salmonella enteritidis* bacteriophages 1, 20, and 11, as well as to other strains of Resistance Groups A, C, and D.

Literature: Burnet, Jour. Path. and Bact., *32*, 1929, 15–42.

22. **Phagus tertius** H. (*loc. cit.*, 154). From Latin *tertius*, third, in reference to the third Resistance Group of *Salmonella enteritidis* bacteriophages, Group C, typified by this bacteriophage.

Common names: *Salmonella enteritidis* bacteriophage 20, 25, 32, 35; Group C bacteriophages.

Hosts: *Salmonella enteritidis* Castellani and Chalmers, *S. gallinarum* Bergey et al., *Shigella dysenteriae* Castellani and Chalmers.

Induced disease: Plaques of small size, with sharp edges.

Immunological relationships: Member of Resistance Group C. Host individuals that have acquired resistance to this bacteriophage are resistant to lines 25, 35, and 32, but susceptible to *Salmonella enteritidis* bacteriophages of Resistance Groups A, B, and D.

Literature: Burnet, Jour. Path. and Bact., *32*, 1929, 15–42.

23. **Phagus dubius** H. (*loc. cit.*, 155). From Latin *dubius*, doubtful, in reference to uncertainty of distinction between Resistance Groups C and D.

Common names: *Salmonella enteritidis* bacteriophage 11, 13; Group D bacteriophages.

Hosts: *Salmonella enteritidis* Castellani and Chalmers, *Shigella dysenteriae* Castellani and Chalmers, *Shigella gallinarum* Weldin.

Induced disease: Very large plaques, up to 8 mm in diameter on 1.2 per cent agar.

Immunological relationships: Member of Resistance Group D. Host individuals that have acquired resistance to this bacteriophage are resistant to line 13, but susceptible to *Salmonella enteritidis* bacteriophages of Resistance Groups A, B, and C.

Literature: Burnet, Jour. Path. and Bact., *32*, 1929, 15–42.

24. **Phagus indicens** *spec. nov.* From Latin *indicere*, to disclose or indicate, in reference to diagnostic use of this bacteriophage in identifying V forms of the typhoid bacillus.

Common name: Phage Q151.

Host: *Salmonella typhosa* White (= *Bacillus typhosus* Zopf).

Insusceptible species: W forms of the typhoid organism and various *Salmonella* species.

Geographical distribution: Canada.

Induced disease: In *Salmonella typhosa*, small plaque formation (lysis) and complete inhibition of growth in cultures of the V form (bearing Vi antigen; resisting O agglutination) and no lysis or restraining effect on growth of the W form (lacking Vi antigen; agglutinated by O antiserum). In the presence of the virus, mixed cultures are quickly transformed since only W variants can increase. Pure V cultures can be identified by the test for their complete inhibition; this inhibition is regularly followed by secondary growth representing the pure W form of the host, a readily formed variant.

Filterability: Passes Seitz EK filter.

Other properties: Filtrates active in dilutions to 10^{-9} or 10^{-11}.

Literature: Craigie, Jour. Bact., *31*, 1936, 56 (Abst.); Craigie and Brandon, Jour. Path. and Bact., *43*, 1936, 233–248, 249–260.

25. **Phagus testabilis** H. (*loc. cit.*, 155). From Latin *testabilis*, able to bear witness, in reference to evidence that this bacteriophage has given, by virtue of its easy destruction when heated in spores, against the hypothesis of frequent spontaneous origin of bacteriophage from the bacterial host.

Common name: *Bacillus megatherium* bacteriophage.

Host: *Bacillus megatherium* De Bary.

Geographical distribution: United States.

Induced disease: Plaques 0.5 mm or less in diameter, with surrounding translucent zone.

Thermal inactivation: *In vitro*, at 75° C in 10 minutes. Spores from infected cultures, after being heated for 10 minutes at 80° C, regularly give rise to subcultures that do not show the presence of this bacteriophage spontaneously during subsequent growth but that are susceptible to lysis if the bacteriophage is again introduced.

Literature: Adant, Compt. rend. Soc. Biol., Paris, *99*, 1928, 1246; Cowles, Jour. Bact., *20*, 1930, 15–23.

26. **Phagus indomitus** H. (*loc. cit.*, 156). From Latin *indomitus*, unrestrained, in reference to the ability of this bacteriophage to increase after heat treatment of infected spores.

Common name: *Bacillus mycoides* bacteriophage.

Host: *Bacillus mycoides* Flügge, some strains.

Insusceptible species: *Bacillus cereus* Frankland and Frankland, *B. subtilis* Cohn emend. Prazmowski, *B. megatherium* De Bary, *B. anthracis* Cohn emend. Koch. Some strains of *B. mycoides*.

Geographical distribution. United States.

Induced disease: Large plaques, with some secondary growth of host organism.

Thermal inactivation: *In vitro*, at 75° C in 10 minutes. Spores from infected cultures, heated at 90° C for 10 minutes give no bacteriophage on grinding, but lytic cultures when grown.

Literature: Lewis and Worley, Jour. Bact., *32*, 1936, 195–198.

27. **Phagus subvertens** H. (*loc. cit.*, 156). From Latin *subvertere*, to subvert, in reference to suspected action of this bacteriophage in causing running-out of alfalfa fields through destruction of nodule organisms.

Common name: *Rhizobium leguminosarum* bacteriophage.

Host: *Rhizobium leguminosarum* Frank. It has been shown that this bacteriophage is unable to increase in clover roots without the nodule-forming organism, *R. leguminosarum*, and that the bacteriophage plays no obviously essential role in nodule formation.

Induced disease: Very small plaques, with edges not sharply defined.

Thermal inactivation: At 60° C in 30 minutes.

Other properties: Not inactivated by drying for 2 months.

Literature: Gerretsen et al., Cent. f. Bakt., II Abt., *60*, 1923, 311–316; Grijns, *ibid.*, *71*, 1927, 248–251; Hitchner, Jour. Bact., *19*, 1930, 191–201; Vandecaveye and Katznelson, Jour. Bact., *31*, 1936, 465–477.

28. **Phagus ineptus** H. (*loc. cit.*, 157). From Latin *ineptus*, unsuitable, in reference to inability of this bacteriophage to adapt itself to lysis of strain RW of its host.

Common name: Streptococcus bacteriophage R.

Host: *Streptococcus cremoris* Orla-Jensen, strain R.

Insusceptible species: *Streptococcus cremoris*, strain RW.

Geographical distribution: New Zealand.

Induced disease: Plaques 0.25 to 0.6 mm in diameter.

Serological relationships: Antisera specific for streptococcus bacteriophage RW

and its strain RW1 are ineffective in neutralizing this bacteriophage.

Immunological relationships: Cultures of host-strain R, after exposure to this bacteriophage, furnish subcultures only partly resistant to this bacteriophage and completely susceptible to streptococcus bacteriophage RW and its substrain RW1.

Literature: Whitehead and Hunter, Jour. Path. and Bact., *44*, 1937, 337–347.

29. **Phagus streptococci** H. (*loc. cit.*, 158). From generic name of host.

Common name: Streptococcus bacteriophage RW.

Host: *Streptococcus cremoris* Orla-Jensen, strain RW.

Geographical distribution: New Zealand.

Induced disease: Plaques 0.25 to 0.6 mm in diameter.

Thermal inactivation: At 70° to 75° C, time not recorded, probably 30 minutes (pH 6.0).

Literature: Whitehead and Hunter, Jour. Path. and Bact., *44*, 1937, 337–347.

Strains: One variant has been described and distinguished from the type variety, *typicus* H. (*loc. cit.*, 158):

29a. *Phagus streptococci* var. *virilis* H. (*loc. cit.*, 158). From Latin *virilis*, vigorous. Common name: Strain RW1 of streptococcus bacteriophage RW. Differing from the type variety in being able to increase at the expense of strain RW1 of *Streptococcus cremoris* (Whitehead and Hunter, Jour. Path. and Bact., *44*, 1937, 337–347).

30. **Phagus maculans** *spec. nov.* From Latin *maculare*, to speckle, in reference to tiny plaques produced by this bacteriophage.

Common name: Streptococcus bacteriophage A.

Hosts: Streptococcus 646, 751, 775.

Geographical distribution: United States (Massachusetts).

Induced disease: Plaques exceedingly minute, scarcely visible to the unaided eye.

Serological relationships: Specific antisera neutralize but there is no cross reaction with respect to streptococcus bacteriophage B, C, or D.

Thermal inactivation: At 60° C in 1 hour.

Other properties: Withstands storage at about 5° C for at least 145 days with but little loss of virulence.

Literature: Evans, Science, *80*, 1934, 40–41; U.S.P.H.S., Public Health Reports, *49*, 1934, 1386–1401.

31. **Phagus lacerans** *spec. nov.* From Latin *lacerare*, to tear, in reference to ragged edges of plaques produced by this bacteriophage.

Common name: Streptococcus bacteriophage B.

Hosts: Streptococcus 563,639: *Streptococcus mucosus* Howard and Perkins.

Insusceptible species: *Streptococcus erysipelatos* Rosenbach.

Geographical distribution: United States (Wisconsin).

Induced disease: Medium size plaques, the largest about 3 mm in diameter, edges ragged, centers clean.

Serological relationships: Specific neutralization, but no cross reactions with streptococcus bacteriophages A, C, and D.

Thermal inactivation: At 60° C in 1 hour.

Other properties: Withstands storage at about 5° C for at least 261 days.

Literature: Clark and Clark, Jour. Bact., *11*, 1926, 89; Proc. Soc. Exp. Biol. and Med., *24*, 1927, 635–639; Colvin, Jour. Inf. Dis., *51*, 1932, 17–29; Evans, U.S.P.H.S., Public Health Reports, *49*, 1934, 1386–1401; Jour. Bact., *39*, 1940, 597–604; Shwartzman, Jour. Exp. Med., *46*, 1927, 497–509.

32. **Phagus tolerans** *spec. nov.* From Latin *tolerans*, tolerating, in reference to the unusual ability of this streptococcus

bacteriophage to remain viable under certain adverse conditions.

Common name: Streptococcus bacteriophage C.

Hosts: Streptococcus 646, 594, 756, 806.

Geographical distribution: United States (Ohio, Massachusetts, Connecticut).

Induced disease: Small plaques, the largest about 1.0 mm in diameter.

Serological relationships: Specific neutralization, but no cross reactions with streptococcus bacteriophages A, B, and D.

Thermal inactivation: At 63° to 65° C in 1 hour.

Other properties: Withstands storage in 1:200 phenol at about 5° C for at least 261 days; equally resistant to storage in 1:10,000 sodium ethyl mercurithiosalicylate (merthiolate), or to storage without preservatives.

Literature: Evans, U. S. Pub. Health Ser., Public Health Reports, *49*, 1934, 1386–1401.

33. **Phagus michiganensis** *spec. nov.* From name of state, Michigan, where this bacteriophage was first isolated.

Common name: Streptococcus bacteriophage D.

Host: Streptococcus 693.

Geographical distribution: United States (Michigan).

Induced disease: Small plaques, about 0.75 mm in diameter, edges clear-cut, centers clean.

Serological relationships: Specific neutralization, but no cross neutralization with streptococcus bacteriophages A, B, and C.

Thermal inactivation: At 60° to 63° C in 1 hour.

Other properties: Withstands storage a t about 5°C for at least 261 days.

Literature: Evans, U. S. Pub. Health Ser., Public Health Reports, *49*, 1934, 1386–1401.

34. **Phagus fragilis**, H. (*loc. cit.*, 159). From Latin *fragilis*, fragile, in reference to easy destruction of this bacteriophage by light and by concentrated urea solutions.

Common names: Staphylococcus bacteriophage Au2, Au3, Au4, or D, perhaps bacteriophage H of Gratia.

Hosts: *Staphylococcus aureus* Rosenbach and *Staphylococcus albus* Rosenbach.

Geographical distribution: United States.

Induced disease: Small plaques, 0.2 to 1.0 mm in diameter, with sharp edges.

Serological relationships: Cross-neutralization reactions with staphylococcus bacteriophages Au1, Au3, Au4, and D, but not with staphylococcus bacteriophages Au21, Au12, A, B, C, or bacteriophage C16.

Thermal inactivation: At about 57° C in 30 minutes.

Other properties: Particle diameter 50 to 75 millimicrons. Readily inactivated photodynamically. Completely inactivated by 27 per cent urea solution in 1 hour at 37° C. Lysis not inhibited even by 1.5 per cent sodium citrate in agar medium.

Literature: Burnet and Lush, Jour. Path. and Bact., *40*, 1935, 455–469; Burnet and McKie, Austral. Jour. Exp. Biol. and Med. Sci., *6*, 1929, 21–31; Fisk, Jour. Inf. Dis., *71*, 1942, 153–160.

35. **Phagus intermedius** H. (*loc. cit.*, 160). From Latin *intermedius*, intermediate, in reference to position of this bacteriophage between staphylococcus bacteriophages that multiply readily in broth cultures of host organisms and those that do not.

Common name: Staphylococcus bacteriophage Au21.

Host: *Staphylococcus aureus* Rosenbach.

Geographical distribution: Australia.

Induced disease: Small plaques, 0.1 to 0.3 mm in diameter, with sharp edges.

Serological relationships: Specific neutralization reaction but no cross-neutralization reaction with staphylococcus bacteriophages Au2 or Au12.

Other properties: Not readily inactivated photodynamically; completely inactivated by 27 per cent urea solution in 1 hour at 37° C; lysis inhibited by 1 per cent sodium citrate in agar medium but not by 0.5 per cent or lower concentrations.

Literature: Burnet and Lush, Jour. Path. and Bact., *40*, 1935, 455–469.

36. **Phagus caducus** H. (*loc. cit.*, 160). From Latin *caducus*, perishable, in reference to the easy destruction of this bacteriophage by concentrated urea solutions.

Common name: Staphylococcus bacteriophage Au12.

Host: *Staphylococcus aureus* Rosenbach.

Geographical distribution: Australia.

Induced disease: Small plaques, 0.2 to 0.5 mm in diameter, with sharp edges.

Serological relationships: Cross-neutralization reactions with staphylococcus bacteriophages Au11 and Au13, but not with staphylococcus bacteriophages Au2, Au21, A, and C. Antiserum to staphylococcus bacteriophage B gives no neutralization of Au12, though the reciprocal reaction occurs to 1:200 dilution.

Other properties: Not readily inactivated photodynamically; completely inactivated by 27 per cent urea solution in 1 hour at 37° C; lysis inhibited by as little as 0.25 per cent sodium citrate in agar.

Literature: Burnet and Lush, Jour. Path. and Bact., *40*, 1935, 455–469.

37. **Phagus alpha** H. (*loc. cit.*, 161). From Greek equivalent of common name.

Common name: Staphylococcus bacteriophage A.

Host: *Staphylococcus albus* Rosenbach.

Geographical distribution: Australia.

Induced disease: Plaques of medium size, 1.5 to 2.5 mm in diameter, with hazy periphery.

Serological relationships: Specific neutralization reaction, but no cross-neutralization reactions with staphylococcus bacteriophages Au2, B, or C.

Immunological relationships: Colonies of *Staphylococcus albus* appearing after lysis with this bacteriophage are resistant to staphylococcus bacteriophages B, C, and D.

Thermal inactivation: At 68° to 70° C in 30 minutes.

Other properties: Not readily inactivated photodynamically; not completely inactivated by 27 per cent urea solution in 1 hour at 37° C; lysis not inhibited even by 1.5 per cent sodium citrate in agar.

Literature: Burnet and Lush, Jour. Path. and Bact., *40*, 1935, 455–469; Burnet and McKie, Austral. Jour. Exp. Biol. and Med. Sci., *6*, 1929, 21–31.

38. **Phagus beta** H. (*loc. cit.*, 162). From Greek equivalent of common name.

Common name: Staphylococcus bacteriophage B.

Host: *Staphylococcus albus* Rosenbach.

Geographical distribution: Australia.

Induced disease: Plaques of medium size, 0.7 to 1.5 mm in diameter, with sharp edges.

Serological relationships: Specific neutralization reaction, but no cross-neutralization reaction with respect to staphylococcus bacteriophages Au2, Au12, A, or C, except that antiserum made with Au12 neutralizes this bacteriophage in low dilutions (See *Phagus caducus*).

Immunological relationships: Colonies appearing after lysis of *Staphylococcus albus* with this bacteriophage furnish organisms susceptible to staphylococcus bacteriophages A and D.

Thermal inactivation: At 63° to 65° C in 10 minutes.

Other properties: Readily inactivated photodynamically; completely inactivated by 27 per cent urea solution in 1 hour at 37° C; lysis not inhibited even by 1.5 per cent sodium citrate in agar medium.

Literature: Burnet and Lush, Jour. Path. and Bact., *40*, 1935, 455–469; Burnet

and McKie, Austral. Jour. Exp. Biol. and Med. Sci., *6*, 1929, 21–31.

39. **Phagus durabilis** H. (*loc. cit.*, 162). From Latin *durabilis*, lasting, in reference to the stability of this bacteriophage in concentrated urea solution and other unfavorable media.

Common name: Staphylococcus bacteriophage C.

Host: *Staphylococcus albus* Rosenbach.

Geographical distribution: Australia.

Induced disease: Plaques 2.0 to 3.0 mm in diameter. Vitreous change in peripheral zone.

Serological relationships: Cross-neutralization reaction with staphylococcus bacteriophage C′, and less strongly with B, but not with Au2 or A.

Immunological relationships: Colonies of *Staphylococcus albus* appearing after lysis with this bacteriophage furnish organisms resistant to it but susceptible to staphylococcus bacteriophages A, B, and D.

Thermal inactivation: At 61° to 63° C in 30 minutes.

Other properties: Not readily inactivated photodynamically; not completely inactivated by 27 per cent urea solution in 1 hour at 37° C; lysis not inhibited even by 1.5 per cent sodium citrate in agar medium.

Literature: Burnet and Lush, Jour. Path. and Bact., *40*, 1935, 455–469; Burnet and McKie, Austral. Jour. Exp. Biol. and Med. Sci., *6*, 1929, 21–31; Rakieten et al., Jour. Bact., *32*, 1936, 505–518.

40. **Phagus liber** H. (*loc. cit.*, 163). From Latin *liber*, independent, in reference to demonstrated independence of this virus, its bacterial host, and its dipterous superhost, in respect to origin.

Common name: *Staphylococcus muscae* bacteriophage.

Host: *Staphylococcus muscae* Glaser.

Geographical distribution: United States.

Induced disease: Lysis in broth cultures; plaques in agar cultures, but characteristics of plaques not recorded.

Thermal inactivation: At a little above 50° C in 5 minutes.

Other properties: A characteristic nucleoprotein has been isolated from lysed staphylococci. Sedimentation constant, 650×10^{-13} cm dyne^{-1} sec.$^{-1}$, corresponding to a molecular weight of about 300,000,000. Denatured at acidities beyond pH 5.0. Digested by chymotrypsin, not by trypsin. Apparent density, about 1.20. Diffusion coefficient, varying with dilution.

Literature: Glaser, Amer. Jour. Hygiene, *27*, 1938, 311–315; Northrop, Jour. Gen. Physiol., *21*, 1938, 335–366; Shope, Jour. Exp. Med., *45*, 1927, 1037–1044; Wyckoff, Jour. Gen. Physiol., *21*, 1938, 367–373.

41. **Phagus cholerae** H. (*loc. cit.*, 164). From former name of host.

Common name: *Vibrio comma* bacteriophage.

Host: *Vibrio comma* Winslow et al. (formerly *V. cholerae* Neisser); Indian strains usually carry this bacteriophage, but Chinese and Japanese strains lack it, are susceptible, and upon inoculation become lysogenic.

Geographical distribution: India.

Induced disease: In both R and S forms of *Vibrio comma*, no plaques on ordinary agar plates, but vibrios become lysogenic. Egg-white in 1:25 dilution enhances activity enough to allow visible lysis, occasional plaques, or stippling at the site of inoculation.

Immunological relationships: *Vibrio comma* organisms that have been infected with this bacteriophage and are resistant to its further action are still susceptible to cholera bacteriophages A, C, and D.

Literature: White, Jour. Path. and Bact., *44*, 1937, 276–278.

42. **Phagus celer** H. (*loc. cit.*, 164). From Latin *celer*, quick, in reference to relatively quick action of this bacteriophage.

Common name: Cholera bacteriophage A.

Host: *Vibrio comma* Winslow et al., smooth types, except non-agglutinable vibrios.

Geographical distribution: India.

Induced disease: Lysis in 2 hours, followed by abundant secondary growth. Only smooth elements of the culture are attacked.

Serological relationships: Antigenically distinct from cholera bacteriophage C.

Immunological relationships: Secondary growth resistant to this virus, but susceptible to cholera bacteriophages C and D.

Other properties: Selectively inactivated by specific polysaccharide of smooth strains, not by a lipoid emulsion that is effective against cholera bacteriophage C. Active in dilution of $1:10^9$ or $1:10^{10}$. Multiplication rate, $n \times 10^5$ in 2 hours.

Literature: Asheshov et al., Indian Jour. Med. Res., *20*, 1933, 1127–1157; White, Jour. Path. and Bact., *43*, 1936, 591–593.

43. **Phagus effrenus** H. (*loc. cit.*, 165). From Latin *effrenus*, unbridled, in reference to the ability of this bacteriophage to attack all tested strains of the cholera organism.

Common name: Cholera bacteriophage C.

Host: *Vibrio comma* Winslow et al., all strains.

Geographical distribution: India.

Induced disease: Sometimes death without lysis. When lysis occurs, it is rarely complete and is followed by secondary resistant growth.

Serological relationships: Antigenically distinct from cholera bacteriophage A.

Immunological relationships: Secondary growth resistant to this bacteriophage, but susceptible to cholera bacteriophages A and D.

Other properties: Selectively inactivated by lipoid from smooth strain of host, but not by specific polysaccharide. Active in dilution of $1:10^8$ or $1:10^{10}$. Multiplication rate, $n \times 10^1$ in 2 hours.

Literature: Asheshov et al., Indian Jour. Med. Res., *20*, 1933, 1127–1157; White, Jour. Path. and Bact., *43*, 1936, 591–593.

44. **Phagus lentus** H. (*loc. cit.*, 166). From Latin *lentus*, slow, in reference to the relatively slow and incomplete lysis induced by this bacteriophage.

Common name: Cholera bacteriophage D.

Host: *Vibrio comma* Winslow et al.

Geographical distribution: India.

Induced disease: Incomplete lysis in about 5 hours, followed, in rough cultures, by slow development of resistant secondary growth.

Immunological relationships: Secondary growth resistant to this bacteriophage, but susceptible to cholera bacteriophages A and C.

Other properties: Not inactivated by specific polysaccharide effective against cholera bacteriophage A, nor by lipoid effective against cholera bacteriophage C. Multiplication rate, $n \times 10^2$ in 2 hours.

Literature: Asheshov et al., Indian Jour. Med. Res., *20*, 1933, 1127–1157; White, Jour. Path. and Bact., *43*, 1936, 591–593.

45. **Phagus diphtheriae** H. (*loc. cit.*, 167). From name of host.

Common name: *Corynebacterium diphtheriae* bacteriophage.

Host: *Corynebacterium diphtheriae* Lehmann and Neumann, many strains, especially 122 of 127 Australian type II *gravis* isolates; type I *gravis* isolates are lysogenic (carriers); all intermediate isolates are susceptible.

Insusceptible species: *Corynebacterium diphtheriae*, all tested *mitis* isolates, except 2 lysogenic. A strain of *C. diphtheriae* from Swan Hill, 200 miles north of Melbourne, was found to be resistant to this bacteriophage and to the small-

plaque diphtheria bacteriophage, *P. futilis*.

Geographical distribution: Australia.

Induced disease: In *Corynebacterium diphtheriae* on agar, plaques 1.0 to 1.5 mm in diameter, with shelving edge. A few resistant bacterial colonies often appear in the central clear area.

Literature: Keogh et al., Jour. Path. and Bact., *46*, 1938, 565–570; Smith and Jordan, Jour. Bact., *21*, 1931, 75–88; Stone and Hobby, Jour. Bact., *27*, 1934, 403–417.

46. **Phagus futilis** H. (*loc. cit.*, 168). From Latin *futilis*, vain, in reference to regular appearance of resistant organisms in plaques on agar cultures lysed by this bacteriophage.

Common name: Small-plaque diphtheria bacteriophage.

Host: *Corynebacterium diphtheriae* Lehmann and Neumann, *gravis* type I isolates and all but 5 *gravis* type II isolates.

Insusceptible species: All tested intermediate and *mitis* strains of *C. diphtheriae*.

Geographical distribution: Australia.

Induced disease: In *Corynebacterium diphtheriae* on agar, pin-point plaques or confluent plaques, with confluent growth of secondary, resistant organisms.

Literature: Keogh et al., Jour. Path. and Bact., *46*, 1938, 565–570.

SUBORDER II. **Phytophagineae** *subordo novus.*

Viruses infecting higher plants; vectors typically homopterous or hemipterous insects (leafhoppers, aphids, white flies, true bugs) or thysanopterous insects (thrips). From Greek *phagein*, to eat, and *phyton*, a plant.

Key to the families of suborder **Phytophagineae.**

1. Inducing yellows-type diseases; vectors typically cicadellid or fulgorid leafhoppers.
   Family I. *Chlorogenaceae*, p. 1145.
2. Inducing mosaic diseases; vectors typically aphids.
   Family II. *Marmoraceae*, p.1163.
3. Inducing ringspot diseases; vectors unknown.
   Family III. *Annulaceae*, p. 1212.
4. Inducing leaf-curl diseases; vectors typically white flies.
   Family IV. *Rugaceae*, p. 1218.
5. Inducing leaf-savoying diseases; vectors, true bugs.
   Family V. *Savoiaceae*, p. 1221.
6. Inducing spotted wilt; vectors, thrips.
   Family VI. *Lethaceae*, p. 1223.

FAMILY I. CHLOROGENACEAE HOLMES EMEND.

(Handb. Phytopath. Viruses, 1939, 1.)

Viruses of the Yellows-Disease Group; pathogenic in flowering plants, causing diseases in which effects on chlorophyll are usually diffuse or stripe-like, no typical spotting or spotty mottling being involved. Vectors, so far as known, leafhoppers (*CICADELLIDAE* and *FULGORIDAE*).

Key to the genera of family **Chlorogenaceae.**

I. True Yellows Group. Viruses inducing diseases usually characterized by stimulation of normally dormant and adventitious buds to produce numerous slender shoots with long internodes and by chlorosis without spotting; invaded parts abnormally erect in habit. Vectors cicadellid leafhoppers so far as known.
   Genus I. *Chlorogenus*, p. 1146.

II. Peach X-Disease Group. Viruses inducing diseases characterized by rosetting of foliage and sometimes death of host.
   Genus II. *Carpophthora*, p. 1151.

III. Phloem-Necrosis Group. Viruses inducing diseases characterized by progressive degeneration of the host plant or by wilting and sudden death; sometimes by root discoloration. Vectors cicadellid leafhoppers so far as known.
   Genus III. *Morsus*, p. 1153.

IV. Yellow-Dwarf Group. Viruses inducing diseases characterized by chlorotic effects somewhat resembling true mottling but often more diffuse. Vectors cicadellid (agallian) leafhoppers.
   Genus IV. *Aureogenus*, p. 1154.

V. Fiji-Disease Group. Viruses inducing diseases characterized by marked vascular proliferation. The vector of one is known to be a leafhopper of the subfamily Delphacinae, family *FULGORIDAE*.
   Genus V. *Galla*, p. 1157.

VI. Stripe-Disease Group. Viruses inducing diseases characterized by chlorotic striping; hosts grasses. Vectors, cicadellid and fulgorid leafhoppers. Genus VI. *Fractilinea*, p. 1159.

Genus I. Chlorogenus *Holmes.*

(*Loc. cit.*, 1.)

Viruses of the Typical Yellows Group, inducing diseases usually characterized by stimulation of normally dormant and adventitious buds to produce numerous slender shoots with long internodes, by chlorosis without spotting, or by both growth of numerous slender shoots and chlorosis. Invaded parts abnormally erect in habit. Affected flowers often virescent. Hosts, dicotyledonous plants. Vectors, so far as known, exclusively cicadellid leafhoppers. Generic name from Greek *chloros*, light green or yellow, and suffix, *gen*, signifying producing, from Greek *genos*, descent.

The type species is *Chlorogenus callistephi* Holmes.

Key to the species of genus **Chlorogenus.**

I. Natural hosts many, in various families of plants.
   1. *Chlorogenus callistephi.*
   2. *Chlorogenus australiensis.*

II. Known natural hosts relatively few.
   A. Natural hosts rosaceous.
      3. *Chlorogenus persicae.*
   B. Natural hosts solanaceous.
      4. *Chlorogenus solani.*
   C. Natural host sandal.
      5. *Chlorogenus santali.*
   D. Natural host cranberry.
      6. *Chlorogenus vaccinii.*
   E. Natural host locust.
      7. *Chlorogenus robiniae.*
   F. Natural host alfalfa.
      8. *Chlorogenus medicaginis.*
   G. Natural host hop.
      9. *Chlorogenus humuli.*

1. **Chlorogenus callistephi** Holmes. (Handb. Phytopath. Viruses, 1939, 2.) From New Latin *Callistephus*, generic name of the China aster.

Common names: Aster-yellows virus, lettuce white-heart virus, *Erigeron*-yellows virus.

Hosts: *Callistephus chinensis* Nees, the China aster, is the host that has been studied most. 170 or more species in 38 different families of dicotyledonous plants have been shown susceptible. Lettuce, endive, carrot, buckwheat, parsnip, and New Zealand spinach are among the hosts of economic importance.

Insusceptible species: All tested species of the family *Leguminosae* and some species of all other tested families have appeared naturally immune.

Geographical distribution: U. S., Canada, Bermuda, Japan, and Hungary. In California the celery-yellows strain of this virus replaces the type.

Induced disease: In most host species the characteristics of disease are clearing of veins, followed by chlorosis of newly formed tissues, stimulation of normally dormant buds to growth, malformation, virescence of flowers, sterility, and upright growth habit. Stimulation of nor-

mally dormant buds to adventitious growth and abnormally erect habit are the most constant features. Chlorosis is absent or inconspicuous in some hosts.

Transmission: By leafhopper, *Macrosteles divisus* (Uhl.) (= *Cicadula sexnotata* (Fall.), *C. divisa* (Uhl.)) (*CICADELLIDAE*). Incubation period, about 2 weeks. Some strains of this virus are transmitted also by the leafhoppers *Thamnotettix montanus* Van D. and *T. geminatus* Van D. (*CICADELLIDAE*). By grafting. By dodder. Not through seeds of diseased plants. Not by mechanical inoculation of plants, but virus has been passed from insect to insect mechanically in *Macrosteles divisus;* juice from viruliferous insects contains little virus just after inoculation, but the effective concentration increases at least 100-fold between the 2nd and 12th day of a 17-day incubation period; it seems greatest before the insects begin to infect the aster plants on which they are maintained.

Thermal inactivation: In juice from viruliferous insects, at about 40° C in 10 minutes; at 25° C in 2 to 3 hours. In plant tissues, at 38° to 42° C, in 2 to 3 weeks; cured plants fully susceptible to reinfection. In insect vector, *M. divisus*, at 31° C in 12 days.

Other properties: Virus in juices derived from insects is more stable at 0° C than at 25° C or when frozen; at 0° C it withstands storage 24, not 48, hours in 0.85 per cent NaCl solution at pH 7.0 but most of the virus is inactivated in this time; it withstands dilution 1:1000 in neutral 0.85 per cent NaCl solution; for brief (less than 5-minute) exposures, it remains viable over the range from pH 5 to 9.

Literature: Black, Phytopath., *31*, 1941, 120–135; *33*, 1943, 2 (Abst.); Johnson, *ibid.*, *31*, 1941, 649–656; Kunkel, Am. Jour. Bot., *13*, 1926, 646–705; Contrib. Boyce Thompson Inst., *3*, 1931, 85–123; *4*, 1932, 405–414; Am. Jour. Bot., *24*, 1937, 316–327; *28*, 1941, 761–769; Linn, Cornell Agr. Exp. Sta. (Ithaca), Bull. 742, 1940; Ogilvie, Bermuda Dept. Agr., Agr. Bull. 6, 1927, 7–8; Severin, Hilgardia, *3*, 1929, 543–583; Phytopath., *20*, 1930, 920–921; Hilgardia, *7*, 1932, 163–179; *8*, 1934, 305–325, 339–361; Phytopath., *30*, 1940, 1049–1051; Hilgardia, *14*, 1942, 411–440; Severin and Haasis, Hilgardia, *8*, 1934, 329–335.

Strains: Two variant strains, one found in nature, the other derived experimentally, have been given varietal names to distinguish them from the type variety, *vulgaris* H. (*loc. cit.*, 2):

1a. *Chlorogenus callistephi* var. *californicus* H. (*loc. cit.*, 3). From California, name of state in which this strain was first recognized. Common name: Celery-yellows strain of aster-yellows virus. Differing from the type variety by ability to infect celery (*Apium graveolens* L.—*UMBELLIFERAE*) and zinnia (*Zinnia elegans* Jacq.—*COMPOSITAE*) (Kunkel, Contrib. Boyce Thompson Inst., *4*, 1932, 405–414; Severin, Hilgardia, *3*, 1929, 543–583; *8*, 1934, 305–325).

1b. *Chlorogenus callistephi* var. *attenuatus* H. (*loc. cit.*, 4). From Latin *attenuatus*, weakened. Common name: Heat-attenuated strain of aster-yellows virus. Differing from the type variety by inducing less severe chlorosis and less uprightness of new growth in affected aster plants (Kunkel, Am. Jour. Bot., *24*, 1937, 316–327).

2. **Chlorogenus australiensis** *comb. nov.* From Australia, name of continent. Synonym: *Galla australiensis* H. (*loc. cit.*, 107).

Common names: Tomato big-bud virus; virescence virus; perhaps also stowboor virus, tobacco stolbur or montar virus, eggplant little-leaf virus.

Hosts: *SOLANACEAE—Datura stramonium* L., Jimson weed; *Lycopersicon esculentum* Mill., tomato; *Nicotiana tabacum* L., tobacco; *Solanum melongena* L., eggplant; *S. nigrum* L., black nightshade. Recently a long list of species in this and

VI. Stripe-Disease Group. Viruses inducing diseases characterized by chlorotic striping; hosts grasses. Vectors, cicadellid and fulgorid leafhoppers. Genus VI. *Fractilinea*, p. 1159.

Genus I. Chlorogenus *Holmes.*

(*Loc. cit.*, 1.)

Viruses of the Typical Yellows Group, inducing diseases usually characterized by stimulation of normally dormant and adventitious buds to produce numerous slender shoots with long internodes, by chlorosis without spotting, or by both growth of numerous slender shoots and chlorosis. Invaded parts abnormally erect in habit. Affected flowers often virescent. Hosts, dicotyledonous plants. Vectors, so far as known, exclusively cicadellid leafhoppers. Generic name from Greek *chloros*, light green or yellow, and suffix, *gen*, signifying producing, from Greek *genos*, descent.

The type species is *Chlorogenus callistephi* Holmes.

Key to the species of genus Chlorogenus.

I. Natural hosts many, in various families of plants.
   1. *Chlorogenus callistephi.*
   2. *Chlorogenus australiensis.*

II. Known natural hosts relatively few.
   A. Natural hosts rosaceous.
      3. *Chlorogenus persicae.*
   B. Natural hosts solanaceous.
      4. *Chlorogenus solani.*
   C. Natural host sandal.
      5. *Chlorogenus santali.*
   D. Natural host cranberry.
      6. *Chlorogenus vaccinii.*
   E. Natural host locust.
      7. *Chlorogenus robiniae.*
   F. Natural host alfalfa.
      8. *Chlorogenus medicaginis.*
   G. Natural host hop.
      9. *Chlorogenus humuli.*

1. **Chlorogenus callistephi** Holmes. (Handb. Phytopath. Viruses, 1939, 2.) From New Latin *Callistephus*, generic name of the China aster.

Common names: Aster-yellows virus, lettuce white-heart virus, *Erigeron*-yellows virus.

Hosts: *Callistephus chinensis* Nees, the China aster, is the host that has been studied most. 170 or more species in 38 different families of dicotyledonous plants have been shown susceptible. Lettuce, endive, carrot, buckwheat, parsnip, and New Zealand spinach are among the hosts of economic importance.

Insusceptible species: All tested species of the family *Leguminosae* and some species of all other tested families have appeared naturally immune.

Geographical distribution: U. S., Canada, Bermuda, Japan, and Hungary. In California the celery-yellows strain of this virus replaces the type.

Induced disease: In most host species the characteristics of disease are clearing of veins, followed by chlorosis of newly formed tissues, stimulation of normally dormant buds to growth, malformation, virescence of flowers, sterility, and upright growth habit. Stimulation of nor-

mally dormant buds to adventitious growth and abnormally erect habit are the most constant features. Chlorosis is absent or inconspicuous in some hosts.

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Thermal inactivation: In juice from viruliferous insects, at about 40° C in 10 minutes; at 25° C in 2 to 3 hours. In plant tissues, at 38° to 42° C, in 2 to 3 weeks; cured plants fully susceptible to reinfection. In insect vector, *M. divisus*, at 31° C in 12 days.

Other properties: Virus in juices derived from insects is more stable at 0° C than at 25° C or when frozen; at 0° C it withstands storage 24, not 48, hours in 0.85 per cent NaCl solution at pH 7.0 but most of the virus is inactivated in this time; it withstands dilution 1:1000 in neutral 0.85 per cent NaCl solution; for brief (less than 5-minute) exposures, it remains viable over the range from pH 5 to 9.

Literature: Black, Phytopath., *31*, 1941, 120–135; *33*, 1943, 2 (Abst.); Johnson, *ibid.*, *31*, 1941, 649–656; Kunkel, Am. Jour. Bot., *13*, 1926, 646–705; Contrib. Boyce Thompson Inst., *3*, 1931, 85–123; *4*, 1932, 405–414; Am. Jour. Bot., *24*, 1937, 316–327; *28*, 1941, 761–769; Linn, Cornell Agr. Exp. Sta. (Ithaca), Bull. 742, 1940; Ogilvie, Bermuda Dept. Agr., Agr. Bull. 6, 1927, 7–8; Severin, Hilgardia, *3*, 1929, 543–583; Phytopath., *20*, 1930, 920–921; Hilgardia, *7*, 1932, 163–179; *8*, 1934, 305–325, 339–361; Phytopath., *30*, 1940, 1049–1051; Hilgardia, *14*, 1942, 411–440; Severin and Haasis, Hilgardia, *8*, 1934, 329–335.

Strains: Two variant strains, one found in nature, the other derived experimentally, have been given varietal names to distinguish them from the type variety, *vulgaris* H. (*loc. cit.*, 2):

1a. *Chlorogenus callistephi* var. *californicus* H. (*loc. cit.*, 3). From California, name of state in which this strain was first recognized. Common name: Celery-yellows strain of aster-yellows virus. Differing from the type variety by ability to infect celery (*Apium graveolens* L.—*UMBELLIFERAE*) and zinnia (*Zinnia elegans* Jacq.—*COMPOSITAE*) (Kunkel, Contrib. Boyce Thompson Inst., *4*, 1932, 405–414; Severin, Hilgardia, *3*, 1929, 543–583; *8*, 1934, 305–325).

1b. *Chlorogenus callistephi* var. *attenuatus* H. (*loc. cit.*, 4). From Latin *attenuatus*, weakened. Common name: Heat-attenuated strain of aster-yellows virus. Differing from the type variety by inducing less severe chlorosis and less uprightness of new growth in affected aster plants (Kunkel, Am. Jour. Bot., *24*, 1937, 316–327).

2. **Chlorogenus australiensis** *comb. nov.* From Australia, name of continent. Synonym: *Galla australiensis* H. (*loc. cit.*, 107).

Common names: Tomato big-bud virus; virescence virus; perhaps also stowboor virus, tobacco stolbur or montar virus, eggplant little-leaf virus.

Hosts: *SOLANACEAE—Datura stramonium* L., Jimson weed; *Lycopersicon esculentum* Mill., tomato; *Nicotiana tabacum* L., tobacco; *Solanum melongena* L., eggplant; *S. nigrum* L., black nightshade. Recently a long list of species in this and

other families have been reported as susceptible to virescence virus, presumed to be an isolate of tomato big-bud virus. (Hill, Jour. Counc. Sci. Ind. Res., *16*, 1943, 85–92).

Geographical distribution: Australia, especially New South Wales; viruses causing somewhat similar diseases have been reported also from the Crimea and the northwestern United States.

Induced disease: In tomato, flowers erect, virescent, calyx bladder-like, pollen sterile; floral proliferation. Growth of axillary shoots stimulated. New leaves progressively smaller. Youngest leaves yellowish-green in color, especially at their margins; usually purplish underneath. Hypertrophy of inner phloem. No intracellular inclusions. Fruit reddens imperfectly and becomes tough and woody. Roots appear normal. In *Solanum nigrum*, axillary shoots numerous, leaves small, internal phloem adventitious. In tobacco, plants dwarfed; leaves recurved, distorted, twisted, thickened, brittle, yellowish green, hanging down close to stem; small leaves on shoots from axillary buds; proliferation and virescence of flowering parts; chlorotic clearing of veins as early effect of disease; upper surface of foliage appears glazed; some necrosis of veins, in old leaves, near tips and margins or on midrib; viable seed rarely produced; calyx bladder-like, floral axis may form short branches bearing small leaves; disease sometimes called bunchy top.

Transmission: By leafhopper, *Thamnotettix argentata* Evans (*CICADELLIDAE*). Experimentally by budding and other methods of grafting. Not by inoculation of expressed juice.

Literature: Cobb, Agr. Gaz. New South Wales, *13*, 1902, 410–414; Dana, Phytopath., *30*, 1940, 866–869; Hill, Jour. Austral. Inst. Agr. Sci., *6*, 1940, 199–200; Jour. Council Sci. Ind. Res., *10*, 1937, 309–312; *16*, 1943, 85–92; Michailowa, Phytopath., *25*, 1935, 539–558; Rischkov et al., Ztschr. Pflanzenkr., *43*, 1933, 496–498; Samuel et al., Phytopath., *23*, 1933, 641–653.

3. **Chlorogenus persicae** H. (*loc. cit.*, 5). From New Latin *Persica*, former generic name of peach.

Common names: Peach-yellows virus, little-peach virus.

Hosts: *ROSACEAE—Prunus persica* (L.) Batsch, peach; *P. salicina* Lindl., Japanese plum; and all other tested species of the genus *Prunus*.

Geographical distribution: Eastern United States and Canada, south to North Carolina. First occurred near Philadelphia in this country. Origin perhaps oriental; introduction in oriental plums suspected. Not in Europe.

Induced disease: In peach, clearing of veins, production of thin erect shoots bearing small chlorotic leaves, followed by death in a year or two. In early stages of the disease there is premature ripening of fruit. In Japanese plum, systemic infection but no obvious symptoms.

Transmission: By the leafhopper, *Macropsis trimaculata* (Fitch) (*CICADELLIDAE*). By budding; virus spreads down stem from point of bud insertion faster than up. Not by inoculation of expressed juice, despite numerous attempts. Not by pollen of diseased trees.

Immunological relationships: Presence of peach-yellows virus immunizes tree against little-peach virus, formerly considered an independent entity.

Thermal inactivation: In peach tissues, at 34° to 35° C in 4 to 5 days; at 44° C in 30 minutes; at 47° C in 10 minutes; at 50° C in 3 to 4 minutes; at 56° C in 15 seconds.

Other properties: Trees and bud sticks may be treated safely with heat sufficient to kill the virus. Cured trees are susceptible to reinfection.

Literature: Blake, N. J. Agr. Exp. Sta., Bull. 226, 1910; Kunkel, Contrib. Boyce Thompson Inst., *5*, 1933, 19–28; Phytopath., *26*, 1936, 201–219, 809–830; *28*, 1938, 491–497; Manns, Trans. Peninsula Hort. Soc., *23*, 1933, 17–19; Manns and Manns,

ibid., *24*, 1934, 72–76; McCubbin, Pennsylvania Dept. Agr., Gen. Bull. *382*, 1924.

Strains: Numerous strains of peach-yellows virus probably exist in nature. One of these has been given a varietal name, distinguishing it from the type variety, *vulgaris* H. (*loc. cit.*, 5):

3a. *Chlorogenus persicae* var. *micropersica* H. (*loc. cit.*, 6). From Greek *micros*, small, and New Latin *Persica*, former generic name of peach. Common name: Little-peach strain of peach-yellows virus. Differing from the type variety by tendency to cause a mild type of disease, characterized by distortion of young leaves, production of many short branches on main trunk, later yellowing of mature leaves, twiggy growth, shoots slightly less erect than in typical peach yellows. (Kunkel, Phytopath., *26*, 1936, 201–219; *26*, 1936, 809–830; *28*, 1938, 491–497; Manns, Trans. Peninsula Hort. Soc., *23*, 1933, 17–19; *24*, 1934, 72–76.)

4. **Chlorogenus solani** H. (*loc. cit.*, 7). From New Latin *Solanum*, generic designation of potato. Synonym: *Chlorophthora solani* McKinney, Jour. Washington Acad. Sci., *34*, 1944, 151.

Common names: Potato witches'-broom virus, potato wilding or semi-wilding virus.

Hosts: *SOLANACEAE—Solanum tuberosum* L., potato. Experimentally, also *SOLANACEAE—Lycopersicon esculentum* Mill., tomato; *Nicotiana tabacum* L., tobacco; *N. glutinosa* L.; *N. rustica* L. *APOCYNACEAE—Vinca rosea* L., periwinkle. *CHENOPODIACEAE—Beta vulgaris* L., sugar beet.

Geographical distribution: United States (Montana, Washington), Russia.

Induced disease: In potato, increasingly pronounced flavescence in new leaflets on one or more stems, production of new dwarfed leaflets with marginal flavescence on stems with unusually long internodes and enlarged nodes, growth of spindling axillary and basal branches, profuse blooming and fruiting, lack of dormancy in tuber buds, formation of many small underground tubers as well as some aerial tubers; plants grown from diseased tubers form thread-like stems and small simple leaves; infected plants survive several seasons, with progressive degeneration. In tomato, experimentally, extreme leaf dwarfing, marginal flavescence of leaves and abnormally numerous axillary branches; stems become hollow and die; plants do not survive long after infection. In tobacco, experimentally, slender axillary branches with dwarfed leaves, flowers on spindling pedicels, numerous, small; later leaves flavescent or marginally flavescent.

Transmission: By tuber-core grafts with prepatent period of 29 to 114 days. By stem grafts. By dodder, *Cuscuta campestris* Yuncker (*CONVOLVULACEAE*). Not by inoculation of expressed juice. Not by *Macrosteles divisus* (Uhl.) (*CICADELLIDAE*). No insect vector is known. Not through seeds of diseased tomatoes.

Thermal inactivation: at 42° C in 13 days, in tissues of *Vinca rosea*; at 36° C in 6 days in small potato tubers.

Literature: Hungerford and Dana, Phytopath., *14*, 1924, 372–383; Kunkel, in Virus Diseases, Cornell Univ. Press, Ithaca, N. Y., 1943, 63–82; Proc. Am. Philosoph. Soc., *86*, 1943, 470–475; McLarty, Scient. Agr., *6*, 1926, 395; Whipple, Montana Agr. Exp. Sta., Bull. 130, 1919; Young, Science, *66*, 1927, 304–306; Am. Jour. Bot., *16*, 1929, 277–279; Young and Morris, Jour. Agr. Res., *36*, 1928, 835–854.

5. **Chlorogenus santali** H. (*loc. cit.*, 8). From New Latin *Santalum*, generic designation of sandal.

Common names: Sandal spike-disease virus, sandal spike-rosette virus.

Hosts: *SANTALACEAE—Santalum album* L., sandal. Spike-like diseases have been found also in *RHAMNEAE—Zizyphus oenoplia* Mill., *SAPINDACEAE—Dodonaea viscosa* Jacq., *VER-*

BENACEAE—Stachytarpheta indica Vahl, and *APOCYNACEAE—Vinca rosea* L.

Geographical distribution: South India.

Induced disease: In sandal, abnormally profuse blooming at first, suppression of blooming later; reduction in leaf size and internode length; death ensues in the third year or earlier. In all but the youngest leaves of affected branches, vacuolate intracellular bodies with definite peripheral membrane, 4 to 9 microns in maximum diameter, are found.

Transmission: By twig grafts, inserted buds, and patch grafts, with success decreasing in the order named. Prepatent period 3 to 4 months. Best results in May and June; poorest in October. Perhaps through seeds, but not through pollen of diseased plants. Insect transmission claimed, but species not identified. Reported transmission by *Moonia albimaculata* (*CICADELLIDAE*) requires further confirmation. Not by inoculation of expressed juice. Not by root grafts.

Literature: Coleman, Mysore Dept. Agr., Mycol. Ser., Bull. 3, 1917; Indian Forester, *49*, 1923. 6-9; Dover, *ibid.*, *60*, 1934, 505-506; Narasimhan, Phytopath., *23*, 1933, 191-202; Rangaswami and Sreenvasaya, Current Science, *4*, 1935, 17-19; Sreenvasaya, Nature, *126*, 1930, 957; Venkata Rao and Gopala Iyengar, Mysore Sandal Spike Invest. Comm., Bull. 4, 1934, 1-12; Indian Forester, *60*, 1934, 689-701.

6. **Chlorogenus vaccinii** H. (*loc. cit.*, 10). From New Latin *Vaccinium*, generic designation of cranberry.

Common names: Cranberry false-blossom virus, Wisconsin false-blossom virus.

Hosts: *ERICACEAE—Vaccinium macrocarpon* Ait., cranberry; *V. oxycoccus* L. Experimentally, also *APOCYNACEAE—Vinca rosea* L., periwinkle. *COMPOSITAE—Calendula officinalis* L., calendula. *SOLANACEAE—Lycopersicon esculentum* Mill., tomato; *Nicotiana glutinosa* L.; *N. tabacum* L., tobacco; *Solanum tuberosum* L., potato.

Geographical distribution: Eastern United States and Canada. It is believed that the virus does not spread in bogs with alkaline (pH 7.4 to 8.8) flooding water in Wisconsin though it spreads rapidly in the more productive bogs with nearly neutral (pH 6.0 to 7.0) flooding water.

Induced disease: In cranberry, flowers erect, instead of pendent as in healthy plants; calyx lobes enlarged, petals short, streaked with red and green, stamens and pistils abnormal. Flowers may be replaced by leaves or short branches. Dormancy of axillary buds is broken, producing numerous erect shoots, forming a witches' broom. Diseased fruits small, irregular in shape, erect.

Transmission: By leafhopper, *Ophiola striatula* (Fall.) (=*Euscelis striatulus* (Fall.)) (*CICADELLIDAE*). Not by inoculation of expressed juice. By dodder, *Cuscuta campestris* Yuncker (*CONVOLVULACEAE*).

Thermal inactivation: At 40° C in 2 weeks in tissues of periwinkle.

Literature: Dobroscky, Contrib. Boyce Thompson Inst., *3*, 1931, 59-83; Fracker, Phytopath., *10*, 1920, 173-175; Kunkel, Science, *95*, 1942, 252; Torreya, *43*, 1943, 87-95; Shear, U. S. Dept. Agr., Bull. 444, 1916; Stevens, Phytopath., *15*, 1925, 85-91; *34*, 1944, 140-142; U. S. Dept. Agr., Circular 147, 1931; Stevens and Sawyer, Phytopath., *16*, 1926, 223-227; Wilcox and Beckwith, Jour. Agr. Res., *47*, 1933, 583-590.

7. **Chlorogenus robiniae** H. (*loc. cit.*, 13). From New Latin *Robinia*, generic designation of locust. Synonym: *Polycladus robiniae* McKinney, Jour. Washington Acad. Sci., *34*, 1944, 151.

Common names: Locust witches'-broom virus; locust brooming-disease virus.

Hosts: *LEGUMINOSAE—Robinia pseudoacacia* L., black locust.

Geographical distribution: United

States (southern Pennsylvania to northeastern Georgia, west to southwestern Ohio and Tennessee).

Induced disease: In black locust, clearing of veins, followed by progressive reduction in size of newly formed leaves; growth of spindly shoots to form witches' brooms. Roots more brittle, shorter, and darker than normal, with excessive branching of rootlets, giving the appearance of root brooms.

Transmission: By grafting and budding. Not by inoculation of expressed juice. No insect vector is known.

Literature: Grant et al., Jour. Forestry, *40*, 1942, 253–260; Hartley and Haasis, Phytopath., *19*, 1929, 163–166; Jackson and Hartley, Phytopath., *23*,1933, 83–90; Waters, Plant World, *1*, 1898, 83–84.

8. **Chlorogenus medicaginis** H. (*loc. cit.*, 14). From New Latin *Medicago*, generic designation of alfalfa (lucerne).

Common names: Alfalfa witches'-broom virus, lucerne witches'-broom virus, spindle-shoot virus, mistletoe virus, Kurrajong virus, bunchy-top virus.

Hosts: *LEGUMINOSAE—Medicago sativa* L., alfalfa (lucerne).

Geographical distribution: Australia, especially New South Wales; perhaps United States.

Induced disease: In alfalfa, plant dwarfed; leaves small, rounded, chlorotic at edge, puckered, distorted; stems short, spindly, numerous. Flowers usually not formed, but sometimes pale and small, sometimes replaced by leafy structures. Seed rarely produced.

Transmission: By grafting. Not by inoculation of expressed juice. No insect vector is known.

Literature: Edwards, Jour. Australian Inst. Agr. Sci., *1*, 1935, 31–32; New South Wales Dept. Agr., Science Bull. 52, 1936; Agr. Gaz. New South Wales, *47*, 1936, 424–426; Richards, U. S. Dept. Agr., Plant Disease Reporter, Supplement, *71*, 1929, 309–310.

9. **Chlorogenus humuli** H. (*loc. cit.*, 15). From New Latin *Humulus*, generic designation of the hop.

Common names: Hop-nettlehead virus, silly-hill disease virus, virus of infectious-sterility of the hop.

Hosts: *MORACEAE—Humulus lupulus* L., European hop.

Geographical distribution: England, Czechoslovakia, Germany, Poland.

Induced disease: In hop, stems numerous, spindly, short, plants weak. Leaves curled upward at margin; cone production greatly reduced.

Transmission: By grafting. Not by inoculation of expressed juice. Not through soil. No insect vector is known.

Literature: Blattný and Vukolov, Rec. Inst. Rech. Agron. Rép. Tchécosl., *137*, 1935, 3–18; Goodwin and Salmon, Jour. Inst. Brew., *33*, 1936, 209–210; Salmon, *ibid.*, *32*, 1935, 235–237; *33*, 1936, 184–186; Salmon and Ware, Jour. South-Eastern Agr. College, Wye, Kent. *37*, 1936, 21–25.

Genus II. **Carpophthora** *McKinney emend.*

(Jour. Washington Acad. Sci., *34*, 1944, 152.)

Peach X-Disease Group; viruses inducing diseases characterized in general by rosetting of foliage and sometimes death of host. Generic name from Greek, meaning fruit and ruin or destruction.

The type species is *Carpophthora lacerans* McKinney.

Key to the species of genus **Carpophthora.**

I. Inducing chlorosis, reddening, and tattering of foliage, with rosette formation late in the disease in some hosts.

1. *Carpophthora lacerans.*

II. Inducing rosette formation characteristically, but not tattering of affected foliage.

2. *Carpophthora rosettae*

1. **Carpophthora lacerans** (Holmes) McKinney. (*Marmor lacerans* Holmes, Handb. Phytopath. Viruses, 1939, 82; McKinney, Jour. Washington Acad. Sci., *34*, 1944, 152.) From Latin *lacerare*, to lacerate, in reference to characteristic foliage injury.

Common name: Peach X-disease virus; virus of peach yellow-red virosis.

Hosts: *ROSACEAE—Prunus persica* (L.) Batsch, peach; *P. virginiana* L., chokecherry.

Geographical distribution: United States, Canada.

Induced disease: In peach, foliage normal each spring but yellowish areas appear in June at base of leaves; affected trees appear lighter green than neighboring healthy trees; discolored spots occur at random on the leaf blade, becoming red and yellow with remainder of leaf becoming chlorotic; the discolored areas usually fall out, leaving the foliage tattered; subsequently, affected leaves drop except at tips of branches; young trees may die, older ones survive indefinitely. Fruit either shrivels and falls or ripens prematurely, with bitter flavor and no viable seed. In chokecherry, conspicuous premature reddening of foliage, development of fruits with dead embryos in the pits; in the second and third seasons after infection, duller colors of foliage, rosettes of small leaves on terminals; death follows the advanced stage of disease.

Transmission: By budding. Not by inoculation of expressed juice. No insect vector has been reported.

Literature: Berkeley, Div. of Botany and Plant Path., Science Service, Dominion Dept. Agr., Ottawa, Canada, Publication *678*, 1941; Boyd, U. S. Dept. Agr., Plant Dis. Rep., *22*, 1938, 334; Hildebrand, Contrib. Boyce Thompson Inst., *11*, 1941, 485–496; Hildebrand and Palmiter, U. S. Dept. Agr., Plant Dis. Rep., *22*, 1938, 394–396; Hildebrand et al., Handbook of virus diseases of stone fruits in North America, Michigan Agr. Exp. Sta., Misc. Publ., 1942, 21–24; Stoddard, Connecticut Agr. Exp. Sta., Circ. *122*, 1938, 54–60; Proc. Connecticut Pomol. Soc., *48*, 1938, 29–32.

2. **Carpophthora rosettae** (Holmes) *comb. nov.* (*Chlorogenus rosettae* H., *nomen nudum*, Phytopath. *29*, 1939, 434; *Nanus rosettae* H., Handb. Phytopath. Viruses, 1939, 125.) From rosette, common name of induced disease, from French, diminutive of *rose*, a rose.

Common name: Peach-rosette virus.

Hosts: *ROSACEAE—Prunus persica* (L.) Batsch, peach; *P. communis* Fritsch, almond; *P. domestica* L., plum. Experimentally, also—*APOCYNACEAE—Vinca rosea* L., periwinkle. *ROSACEAE—P. americana* Marsh., wild plum; *P. armeniaca* L., apricot; *P. cerasus* L., cherry; *P. pumila* L., sand cherry. *SOLANACEAE—Lycopersicon esculentum* Mill., tomato; *Nicotiana glutinosa* L.

Geographical distribution: United States (Georgia, Alabama, South Carolina, Tennessee, West Virginia, Missouri, Oklahoma).

Induced disease: In peach, sudden wilting and death, or growth of abnormally short stems bearing dwarfed leaves with clearing and thickening of veins, followed by death in a few months.

Transmission: By budding. By dodder, *Cuscuta campestris* Yuncker. Not by inoculation of expressed juice. Not through soil. No insect vector is known.

Immunological relationships: No protection is afforded by previous infection of peach trees with *Chlorogenus persicae*, peach-yellows virus.

Thermal inactivation: At 50° C in 10 minutes (in tissues of peach). Rosetted trees are abnormally susceptible to heat

injury and heat treatments cure peach-rosette disease only in recently infected trees.

Literature: Kunkel, Phytopath., *26*, 1936, 201–219, 809–830; in Virus Diseases, Cornell Univ. Press, Ithaca, N. Y., 1943, 63–82; McClintock, Jour. Agr. Res., *24*, 1923, 307–316; Phytopath., *21*, 1931, 373–386; Smith, U. S. Dept. Agr., Div. Veg. Path., Bull. 1, 1891.

Genus III. **Morsus** *gen. nov.*

Alfalfa-Dwarf Group; viruses inducing diseases characterized in general by sudden wilting and death or by gradual decline of vigor with foliage of darker green color than normal. Vectors, like those of the typical yellows subgroup, cicadellid leafhoppers so far as known. Generic name from Latin *morsus*, sting or vexation.

The type species is *Morsus suffodiens spec. nov.*

Key to the species of genus **Morsus.**

I. Affecting alfalfa and grape. 1. *Morsus suffodiens.*

II. Affecting tobacco. 2. *Morsus reprimens.*

III. Affecting elm. 3. *Morsus ulmi.*

1. **Morsus suffodiens** *spec. nov.* From Latin *suffodere*, to sap or undermine, in reference to process leading to sudden collapse of long infected, but sometimes not obviously injured, grape vines as well as to progressive decline in size of infected alfalfa plants, the foliage of which may remain green to the last.

Common names: Alfalfa-dwarf virus, lucerne dwarf-disease virus, virus of Pierce's disease of the grape, virus of Anaheim disease.

Hosts: *LEGUMINOSAE—Medicago sativa* L., alfalfa (lucerne). *VITACEAE—Vitis vinifera* L., grape.

Geographical distribution: United States.

Induced disease: In alfalfa, leaves small but green, plant progressively smaller, wood of roots discolored yellow, transpiration decreased; wilting may occur; starch of root diminished; plant eventually succumbs, thinning stand prematurely. In grape, dark green color of leaves retained along veins, not between them, or no abnormality in appearance of foliage; wilting and sudden death of plant in summer of second year. In late summer of first year, there may be dying leaf margins and dying back of cane tips.

Transmission: By budding and root grafting. By leafhoppers, *Draeculacephala minerva* Ball, *Carneocephala fulgida* Nott., *C. triguttata* Nott., *Helochara delta* Oman, *Neokolla circellata* (Baker), *N. gothica* (Sign.), *N. confluens* (Uhler), *N. heiroglyphica* (Say), and *Cuerna occidentalis* Oman and Beamer (*CICADELLIDAE*); these vectors all belong to the subfamily *Amblycephalinae;* all tested species of this, but none of any other, subfamily have proved capable of transmitting this virus. Not by inoculation of expressed juice. Not through soil.

Literature: Frazier, Phytopath., *34*, 1944, 1000–1001; Hewitt, Phytopath., *29*, 1939, 10; *31*, 1941, 862; Blue Anchor, *18*, 1941, 16–21, 36; Hewitt et al., Phytopath., *32*, 1942, 8; Houston et al., Phytopath., *32*, 1942, 10; Milbrath, Calif. Dept. Agr., 20th Ann. Rept., Bull. *28*, 1940, 571; Pierce, U. S. Dept. of Agr., Div. of Veg. Path., Bull. *2*, 1892, 1–222; Weimer, Phytopath., *21*, 1931, 71–75; *27*, 1937, 697–702; Jour. Agr. Res., *47*, 1933, 351–368; *53*, 1936, 333–347; *55*, 1937, 87–104.

2. **Morsus reprimens** *spec. nov.* From Latin *reprimere*, to restrain, check, or curb, in reference to the inhibiting effect on growth of the host plant, tobacco.

Common name: Tobacco yellow-dwarf virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., tobacco; *N. rustica* L., Indian tobacco; *N. trigonophylla* Dun. Experimentally, also *N. glauca* Grah. (symptomless) and *N. glutinosa* L.

Geographical distribution: Australia (Victoria, New South Wales, South Australia, and southern Queensland).

Induced disease: In tobacco, internodes of stem shortened, leaves small; downward bending of tips and rolling under of margins of young apical leaves; young leaves darker than normal at first, bunched, later appear ribbed; leaves become yellow-green, pale first between veins; old leaves rugose, thickened, later savoyed. Root system small, roots slightly brown externally and in the region of the phloem. Affected plants may survive the winter and show diseased new growth in the spring.

Transmission: By grafting and budding. By nymphs and adults of the leafhopper, *Thamnotettix argentata* (Evans) (*CICADELLIDAE*).

Literature: Dickson, Australia, Council Sci. Indust. Res., Pamphlet 14, 1929, 22; Hill, Australia, Journal of the Council Sci. Indust. Res., *10*, 1937, 228–230; *14*, 1941, 181–186; *15*, 1942, 13–25.

3. **Morsus ulmi** *spec. nov.* From Latin *ulmus*, elm.

Common name: Elm phloem-necrosis virus.

Host: *URTICACEAE—Ulmus americana* L., American elm.

Geographical distribution: United States (Ohio, Indiana, Illinois, Missouri, Tennessee, Kentucky, and West Virginia).

Induced disease: In elm, gradual decline over a period of 12 to 18 months before death or sudden wilt, drying of leaves, and death within 3 to 4 weeks. All ages susceptible, from seedling to large tree.

Transmission: By patch grafting. Not by inoculation of expressed juice.

Literature: Leach and Valleau, U. S. Dept. Agr., Plant Dis. Rept., *23*, 1939, 300–301; Swingle, Phytopath., *30*, 1940, 23.

Genus IV. Aureogenus *Black.*

(Proc. Am. Philos. Soc., *88*, 1944, 141.)

Viruses of the Yellow-Dwarf Group, inducing diseases characterized by yellowing without typical mosaic-type mottling. Vectors agallian leafhoppers (*CICADELLIDAE*). Generic name from Latin *aureus*, yellow or golden, and *genus*, group.

The type species is *Aureogenus vastans* (Holmes) Black.

Key to the species of genus Aureogenus.

I. Mechanically transmissible in some hosts by rubbing methods of inoculation. Not producing enlarged veins or club-leaf in clover.
1. *Aureogenus vastans.*

II. Not known to be transmissible by rubbing methods of inoculation.
A. Producing enlarged veins in clover.
2. *Aureogenus magnivena.*
B. Producing club-leaf in clover.
3. *Aureogenus clavifolium.*

1. **Aureogenus vastans** (Holmes) Black. (*Marmor vastans* Holmes, Handb. Phytopath. Viruses, 1939, 94; Black, Proc. Am. Philos. Soc., *88*, 1944, 141.) From Latin *vastare*, to devastate.

Common name: Potato yellow-dwarf virus.

Hosts: *SOLANACEAE—Solanum tuberosum* L., potato. *COMPOSITAE—Chrysanthemum leucanthemum* L., var. *pinnatifidum* Lecoq and Lamotte, daisy; *Rudbeckia hirta* L., black-eyed Susan. *CRUCIFERAE—Barbarea vulgaris* R. Br., ommon winter cress. *LEGUMINOSAE—Trifolium pratense* L., red clover. Experimentally to numerous species in these and other families.

Geographical distribution: Northeastern United States and southeastern Canada.

Induced disease: In potato, yellowing of leaves, necrosis of stem, dwarfing of plant; the stem, if split, shows rusty specks especially at nodes and apex; the apex dies early; tubers are few, small, close to the stem, often cracked, with flesh discolored by scattered brown specks; seed tubers tend to remain unrotted in the ground, becoming hard and glassy; some of them do not germinate in warm soil, others produce shoots that die before reaching the surface, giving poor stands. In *Chrysanthemum leucanthemum* var. *pinnatifidum*, at first, clearing of veins; later, young leaves distorted, thick, stiff, small; petioles short, leaves erect, forming a rosette at the crown of the plant; with passing of the early phases of the disease, foliage tends to appear nearly normal, but remains darker green and more erect than that of healthy plants; virus is recoverable both during and after the period of obvious disease and infected plants may constitute an important reservoir. In *Trifolium incarnatum* L., crimson clover, experimentally, clearing of veins and yellowing of younger leaves (in summer the yellowing is usually replaced in part by an interveinal reddish-brown color on both leaf surfaces extending from the margins inwards); dwarfing of entire plant; death or a chronic disease characterized by milder manifestations without, however, vein enlargement or cupping of leaves. In *Nicotiana rustica* L., experimentally, yellowish primary lesions followed by clearing of veins and systemic chlorosis; the primary lesions facilitate quantitative estimation of concentrations of this virus.

Transmission: By inoculation of expressed juice, in the presence of finely powdered carborundum, to *Nicotiana rustica*; mechanical transmission very difficult in other hosts tested. By grafting. By clover leafhopper, *Aceratagallia sanguinolenta* (Provancher); experimentally, by other closely related leafhoppers, *Aceratagallia lyrata* (Baker), *A. obscura* Oman, and *A. curvata* Oman; not (for the type variety of the virus) by *Agallia constricta* Van Duzee; very rarely by *Agallia quadripunctata* (Provancher) and *Agalliopsis novella* (Say) (*CICADELLIDAE*). The vector *Aceratagallia sanguinolenta* remains infective as an overwintering adult; incubation period not less than 6 days, commonly much longer; virus does not pass to progeny of viruliferous leafhoppers through eggs or sperm; this leafhopper varies genetically in ability to transmit.

Immunological relationships: No protection is afforded against necrotic effects of a testing strain of this virus (var. *lethale* Black) by prior inoculation of *Nicotiana rustica* with isolates of *Marmor medicaginis*, *M. cucumeris*, *M. upsilon*, *Annulus tabaci*, *A. orae*, or *A. dubius*, but the varieties *vulgare* Black and *agalliae* Black protect; these specifically protecting strains give no similar protection against formation of necrotic lesions by subsequently applied isolates of *Marmor tabaci*, *M. lethale*, *Annulus tabaci*, or *A. orae*.

Thermal inactivation: At 50 to 52° C in 10 minutes.

Filterability: Passes Berkefeld W filter.

Other properties: Virus viable at 23 to 27° C less than 13 hours after extraction of juice from diseased plant; not infective after drying in leaf tissues.

Literature: Barrus and Chupp, Phytopath., *12*, 1922, 123–132; Black, Am. Potato Jour., *11*, 1934, 148–152; Cornell Univ. Agr. Exp. Sta., Mem. 209, 1937, 1–23; Phytopath., *28*, 1938, 863–874; Am. Jour. Bot., *27*, 1940, 386–392; Am. Potato Jour., *18*, 1941, 231–233; Phytopath., *33*, 1943, 363–371; Genetics, *28*, 1943, 200–209; Proc. Am. Philos. Soc., *88*, 1944, 132–144; Hansing, Cornell Univ. Agr. Exp. Sta., Bull. 792, 1943; Price and Black, Am. Jour. Bot., *28*, 1941, 594–595; Taylor, Am. Potato Jour., *15*, 1938, 37–40; Walker and Larson, Jour. Agr. Res., *59*, 1939, 259–280; Watkins, Jour. Econ. Ent., *32*, 1939, 561–564; Cornell Univ. Agr. Exp. Sta., Bull. 758, 1941, 1–24; Younkin, Am. Potato Jour., *19*, 1942, 6–11.

Strains: Beside the type variety, *Aureogenus vastans* var. *vulgare* Black (Am. Jour. Bot., *27*, 1940, 391), on which the species is based, two distinctive strains have been given varietal names:

1a. *Aureogenus vastans* var. *agalliae* Black. (Am. Potato Jour., *18*, 1941, 233.) From New Latin *Agallia*, generic name of vector of this strain. Common name: New Jersey strain of potato yellow-dwarf virus. Differing from the type especially in its distinctive vector, the leafhopper, *Agallia constricta* Van Duzee, which is incapable of transmitting the type strain, and in not being transmitted by *Aceratagallia sanguinolenta* (Provancher), common vector of the type variety. Experimentally, transmitted also by *Agallia quadripunctata* (Provancher); perhaps rarely by *Agalliopsis novella* (Say); Differing but little from the type in effects on potato (var. Green Mountain) and *Nicotiana rustica* but more definitely in effects on crimson clover, in affected plants of which a rusty-brown necrosis along the veins, not induced by the type strain, is always present in some degree.

1b. *Aureogenus vastans* var. *lethale* Black. (Am. Jour. Bot., *27*, 1940, 391.) From Latin *lethalis*, causing death. Common name: Strain B5 of potato yellow-dwarf virus. Differing from the type variety especially in a tendency to induce in *Nicotiana rustica*, experimentally, brown primary lesions with necrotic gray centers, systemic yellowing, extensive necrosis of veins, collapse of large areas of leaf, and sometimes death of the host; not known to occur in nature as a separate strain, but readily isolated as a variant from strains collected in nature.

2. **Aureogenus magnivena** Black. (Proc. Am. Philos. Soc., *88*, 1944, 144.) From Latin *magnus*, large, and *vena*, vein.

Common name: Clover big-vein virus.

Host: Experimentally, *LEGUMINOSAE—Trifolium incarnatum* L., crimson clover.

Insusceptible species: *SOLANACEAE—Nicotiana rustica* L., Indian tobacco; *Solanum tuberosum* L., potato.

Geographical distribution: United States (presumably, Washington, D. C.).

Induced disease: In crimson clover, experimentally, unevenly thickened veins which are depressed below the upper surface of the leaf; these enlarged veins, best observed from below, sometimes bear enations that arise from their lower surfaces, leaves often curl upward and inward marginally; in summer, yellowing of leaves progresses from margins inward, the yellow color being later replaced in part by red or purple red; petioles undulating; plants dwarfed; internodes shortened; no clearing of veins; no rusty-brown necrosis.

Transmission: Not by inoculation of expressed juice. By leafhoppers, *Agalliopsis novella* (Say), *Agallia constricta* Van Duzee, *A. quadripunctata* (Provancher); not by *Aceratagallia sanguinolenta* (Provancher) (*CICADELLIDAE*).

3. **Aureogenus clavifolium** Black. (Proc. Am. Philos. Soc., *88*, 1944, 141.) From Latin *clava*, club, and *folium*, leaf.

Common name: Clover club-leaf virus.

Host: Experimentally, *LEGUMINOSAE—Trifolium incarnatum* L., crimson clover.

Insusceptible species: *SOLANACEAE—Nicotiana rustica* L., Indian tobacco; *Solanum tuberosum* L., potato.

Geographical distribution: United States (Princeton, N. J.).

Induced disease: In crimson clover, experimentally, youngest leaves lighter green than normal, slow to unfold; leaf margins yellowed or colored red or purple red; affected leaves narrow, smooth or savoyed; plant dwarfed, new shoots from leaf axils slightly stimulated; new growth of spindly stems and small leaves; no rusty-brown necrosis of veins, no obvious enlargement of veins, and no obvious clearing of veins at the onset of disease.

Transmission: Not by inoculation of expressed juice. By leafhopper, *Agalliopsis novella* (Say) (*CICADELLIDAE*); not by leafhoppers, *Aceratagallia sanguinolenta* (Provancher), *Agallia constricta* Van Duzee, nor *A. quadripunctata* (Provancher) (*CICADELLIDAE*).

Genus V. **Galla** *Holmes.*

(*Loc. cit.*, 106)

Viruses of the Fiji-Disease Group, inducing diseases characterized by vascular proliferation. Generic name from Latin *galla*, a gall nut.

The type species is *Galla fijiensis* Holmes.

Key to the species of genus **Galla.**

I. Infecting sugar cane.
  A. Inducing formation of conspicuous galls.
    1. *Galla fijiensis.*
  B. Not inducing formation of conspicuous galls.
    2. *Galla queenslandiensis.*
II. Infecting anemone.
    3. *Galla anemones.*
III. Infecting peach.
    4. *Galla verrucae.*
IV. Infecting corn.
    5. *Galla zeae.*

1. **Galla fijiensis** Holmes. (Handb. Phytopath. Viruses, 1939, 106.) From name of Fiji Islands.

Common name: Fiji-disease virus.

Host: *GRAMINEAE—Saccharum officinarum* L., sugar cane.

Geographical distribution: Fiji Islands, New South Wales, Java, Philippine Islands, New Guinea and New Caledonia.

Induced disease: In sugar cane, galls on vascular bundles, formed by proliferation of phloem and nearby cells. Affected cells show characteristic spherical or oval inclusion bodies. Developing leaves shortened, crumpled, abnormally dark green. Infected stools of cane become bushy. Roots small, bunchy.

Transmission: By leafhoppers, *Perkinsiella saccharicida* Kirk. (in Queensland) and *P. vastatrix* Breddin (in Philippine Islands) (*FULGORIDAE*, subfamily *Delphacinae*). Not by grafting. Not by inoculation of expressed juice. Not through eggs of *P. vastatrix*. Cuttings taken from affected canes produce some healthy and some diseased plants, because virus does not become uniformly distributed throughout the host tissues.

Literature: Kunkel, Bull. Exp. Sta., Hawaiian Sugar Planters' Assoc., Bot.

Ser., *3*, 1924, 99–107; Lyon, *ibid.*, *3*, 1921, 1–43; Hawaiian Planters' Rec., *12*, 1915, 200; Mungomery and Bell, Queensland, Bur. Sugar Exp. Sta., Div. Path., Bull. 4, 1933; Ocfemia, Am. Jour. Bot., *21*, 1934, 113–120; Ocfemia and Celino, Phytopath., *29*, 1939, 512–517; Reinking, Phytopath., *11*, 1921, 334–337.

2. **Galla queenslandiensis** H. (*loc. cit.*, 109). From Queensland, where the induced disease was first studied.

Common name: Sugar-cane dwarf-disease virus.

Host: *GRAMINEAE—Saccharum officinarum* L., sugar cane.

Geographical distribution: Queensland.

Induced disease: In sugar cane, young leaves marked with scattered chlorotic streaks, leaves stiff and erect, spindle twisted, abnormally short and pale. As leaves mature, streaks disappear, leaves become darker than normal green. In recently infected plants, vascular bundles are enlarged, irregular in shape, fused and characterized by abnormal proliferation of thin-walled lignified cells.

Literature: Bell, Queensland, Bur. Sugar Exp. Sta., Div. Path., Bull. 3, 1932.

3. **Galla anemones** H. (*loc. cit.*, 108). From Latin *anemone*, anemone or windflower.

Common name: Anemone-alloiophylly virus.

Hosts: *RANUNCULACEAE—Anemone nemorosa* L., vernal windflower; *A. ranunculoides* L.; *A. trifolia* L.

Geographical distribution: Germany.

Induced disease: Leaves thickened and distorted, petioles thickened. Flowers distorted or not formed. Vascular bundles larger and more numerous than in healthy plants. Palisade cells short, chloroplasts smaller and fewer than normal.

Transmission: By needle puncture into rhizomes immersed in filtered juice of diseased plant. By contamination of soil with fragments of diseased leaves or rhizomes.

Literature: Klebahn, Bericht. d. Deutsch. Bot. Gesellsch., *15*, 1897, 527–536; Ztschr. wissensch. Biol., Abt. E, Planta, *1*, 1926, 419–440; *6*, 1928, 40–95; Phytopath. Ztschr., *4*, 1932, 1–36; *9*, 1936, 357–370.

4. **Galla verrucae** Blodgett. (Phytopath., *33*, 1943, 30.) From Latin *verruca*, wart. Originally spelled *verruca*, apparently by a typographical error, which was corrected twice on the following page, once in a statement that the genitive *verrucae* had been given as specific epithet.

Common name: Peach-wart virus.

Host: *ROSACEAE—Prunus persica* (L.) Batsch, peach.

Geographical distribution: United States (Idaho, Washington, Oregon).

Induced disease: In peach, no characteristic effect on foliage. Fruits blistered, welted, later with warty outgrowths conspicuously raised. Affected tissues light tan to red, rough, cracked and russeted, or smooth. Gumming usual, often severe. Warty tissue superficial; underlying tissues coarse, filled with gum pockets, but not abnormal in flavor. Warty tissue may be hard and bony, but more often it is merely tougher than normal.

Transmission: By budding and inarching.

Literature: Blodgett, Phytopath., *31*, 1941, 859–860 (Abst.); *33*, 1943, 21–32.

5. **Galla zeae** McKinney. (Jour. Washington Acad. Sci., *34*, 1944, 328.) From Latin *zea*, a kind of grain.

Common name: Wallaby-ear disease virus.

Host: *GRAMINEAE—Zea mays* L., corn (maize).

Geographical distribution: Southeastern Queensland, Australia.

Induced disease: In corn (maize), small swellings on secondary veins on undersides of young leaves, spreading to base and tip of leaf along veins; plant dwarfed, becoming abnormally deep green and deficient in development of pollen; silk, cobs, and grain retarded in growth.

Transmission: By leafhopper, *Cicadula bimaculata* Evans (*CICADELLIDAE*). Literature: Schindler, Jour. Austral. Inst. Agr. Sci., *8*, 1942, 35–37.

Genus VI. **Fractilinea** *McKinney.*

(Jour. Washington Acad. Sci., *34*, 1944, 148.)

Viruses of the Stripe-Disease Group; hosts grasses; insect vectors, cicadellid and fulgorid leafhoppers. Generic name from Latin, meaning interrupted and line.

The type species is *Fractilinea maidis* (Holmes) McKinney.

Key to the species of genus **Fractilinea.**

I. Vectors, cicadellid leafhoppers.

1. *Fractilinea maidis.*
2. *Fractilinea oryzae.*
3. *Fractilinea tritici.*
4. *Fractilinea quarta.*

II. Vectors, fulgorid leafhoppers.

5. *Fractilinea zeae.*
6. *Fractilinea avenae.*

1. **Fractilinea maidis** (Holmes) McKinney. (*Marmor maidis* Holmes, Handb. Phytopath. Viruses, 1939, 56; *Fractilinea maidis* McKinney, Jour. Washington Acad. Sci., *34*, 1944, 149.) From New Latin *mays*, corn (i.e. maize).

Common name: Maize-streak virus.

Hosts: *GRAMINEAE—Zea mays* L., corn (maize); *Digitaria horizontalis* Willd., *Eleusine indica* Gaert.; *Saccharum officinarum* L., sugar cane.

Geographical distribution: Africa.

Induced disease: In corn, pale spots at base of young leaf, followed by chlorotic spotting and streaking of subsequently formed leaves. Virus moves rapidly (up to 40 cm in 2 hours at 30° C) after introduction into host plant by insect. More virus in chlorotic spots than in green areas of affected leaves.

Transmission: By leafhoppers, *Cicadulina* (= *Balclutha*) *mbila* (Naude), *C. zeae* China, and *C. storeyi* China (*CICADELLIDAE*). In *C. mbila* ability to transmit this virus is controlled by a sex-linked dominant gene; active male (AX) (Y), inactive male (aX) (Y), inactive female (aX) (aX), active female (AX) (AX) or (AX) (aX). Inactive individuals ingest virus when feeding, but can become infective only if the intestine is wounded purposely or accidentally. If inoculated artificially by introducing virus into blood, both active and inactive insects become infective. Incubation period, 6 to 12 hours at 30° C. Young not infected through the egg. Infective leafhopper cannot transmit virus unless feeding puncture exceeds a minimum period, about 5 minutes in duration. This virus has not been transmitted to its plant hosts by inoculation of expressed juices.

Filterability: At pH 6, passes Chamberland L_1 and L_3, Berkefeld V and N, filters; retained by Seitz E K filter disc.

Literature: Storey, Ann. Appl. Biol., *12*, 1925, 422–439; *15*, 1928, 1–25; *19*, 1932, 1–5; Proc. Roy. Soc., B, *112*, 1932, 46–60; *113*, 1933, 463–485; *125*, 1938, 455–477; Ann. Appl. Biol., *21*, 1934, 588–589; *24*, 1937, 87–94; East Afr. Agr. Jour., *1*, 1936, 471–475; Storey and McClean, Ann. Appl. Biol., *17*, 1930, 691–719.

Strains: Two strains that differ radically from the type, var. *typicum* H. (*loc. cit.*, 56), have been given varietal names, as follows:

1a. *Fractilinea maidis* var. *sacchari* H. (*loc. cit.*, 57). From New Latin *Saccharum*, generic name of sugar cane. Common name: Cane-streak strain of

maize-streak virus. Differing from the type strain in being specialized for attacking sugar cane, in which the type (from maize) tends to be localized or finally lost with resultant spontaneous recovery of the temporary host. The cane-streak strain usually spreads readily in the cane plant; leaves become much marked with broken, narrow, pale, longitudinal stripes and spots; stems remain unaffected. One variety of sugar cane, P.O.J. 213, is resistant and, if infected, tends to recover. (McClean, Intern. Soc. Sugar Cane Techn., Bull. 27, 1932; Proc. So. Afr. Sugar Techn. Assoc., 1936, 1–11; Storey, Rept. Imp. Bot. Conf., London, 1924, 132–144; Union So. Afr. Dept. Agr., Sci. Bull. 39, 1935; Ann. Appl. Biol., *17*, 1930, 691–719.

1b. *Fractilinea maidis* var. *mitis* H. (*loc. cit.*, 58). From Latin *mitis*, mild. Common name: Mottle strain of maize-streak virus. Differing from the typical strain by the mildness of the disease induced in corn (maize), transitory chlorotic mottling of newly developed leaves, followed by fading of mottling and production of apparently normal leaves. Young leaves, while mottled, are less rigid than normal and may not remain as nearly erect as healthy leaves. (Storey, Ann. Appl. Biol., *24*, 1937, 87–94.)

2. **Fractilinea oryzae** (Holmes) *comb. nov.* (*Marmor oryzae* Holmes, *loc. cit.*, 64.) From Latin *oryza*, rice.

Common name: Rice dwarf-disease virus.

Hosts: *GRAMINEAE—Oryza sativa* L., rice. Experimentally, also *Alopecurus fulvus* L.; *Avena sativa* L., oat; *Echinochloa crusgalli* Beauv. var. *edulis* Honda; *Panicum miliaceum* L.; *Poa pratensis* L.; *Secale cereale* L., rye; *Triticum vulgare* Vill., wheat.

Insusceptible species: *GRAMINEAE —Zea mays* L., corn (maize); *Hordeum vulgare* L., barley; *Setaria italica* Beauv., foxtail millet; *Andropogon sorghum* Brot. (= *Holcus sorghum* L.), sorghum.

Geographical distribution: Japan, Philippine Islands.

Induced disease: In rice, yellowish green spots along veins of young leaf, followed by chlorotic spotting and streaking of subsequently formed leaves. Growth stunted, internodes and roots abnormally short, forming a dwarf plant. Vacuolate intracellular bodies, 3 to 10 by 2.5 to 8.5 microns in size, close to nuclei of cells in affected tissues.

Transmission: By leafhoppers, *Nephotettix apicalis* var. *cincticeps* Uhler, *N. bipunctatus* Fabr., and *Deltocephalus dorsalis* Motsch. (*CICADELLIDAE*). Virus transmitted through some of the eggs but through none of the sperm of infected individuals of *N. apicalis*. Transfer from individuals thus infected through the egg to their progeny likewise demonstrated, even to the 7th generation. This is the only confirmed instance of transmission of a phytopathogenic virus through the eggs of an insect vector and is considered as evidence that the virus multiplies within the body of its vector as well as in its plant hosts. Incubation period in insect usually 30 to 45 days after first feeding on an infected plant, sometimes as short as 10 or as long as 73 days; nymphs from viruliferous eggs do not become infective until 7 to 38 (average 19) days after emergence. Transmission by inoculation of expressed juice has not been demonstrated. No transmission through seeds from diseased rice plants. No soil transmission.

Literature: Agati et al., Philippine Jour. Agr., *12*, 1941, 197–210; Fukushi, Trans. Sapporo Nat. Hist. Soc., *12*, 1931, 35–41; Proc. Imp. Acad., Tokyo, *9*, 1933, 457–460; Jour. Fac. Agr. Hokkaido Imp. Univ., *37*, 1934, 41–164; Trans. Sapporo Nat. Hist. Soc., *13*, 1934, 162–166; Proc. Imp. Acad., Tokyo, *11*, 1935, 301–303; *13*, 1937, 328–331; *15*, 1939, 142–145; Jour. Fac. Agr. Hokkaido Imp. Univ., *45*, 1940, 83–154; Katsura, Phytopath.,

26, 1936, 887–895; Takata, Jour. Japan Agr. Soc., *171*, 1895, 1–4; *172*, 1896, 13–32 (Takata's papers, in Japanese, constitute the first published record of transmission by an insect of a virus causing disease in a plant, the leafhopper *Deltocephalus dorsalis* Motsch. transmitting dwarf-disease virus to rice; see Fukushi, 1937, cited above).

3. **Fractilinea tritici** McKinney. (Jour. Washington Acad Sci., *34*, 1944, 327.) From Latin *triticum*, wheat.

Common name: Winter-wheat mosaic virus.

Hosts: *GRAMINEAE—Triticum aestivum* L., wheat; *Secale cereale* L., rye; *Avena byzantina*; *A. fatua* L., wild oat; *A. sativa* L., oat; *Hordeum vulgare* L., barley.

Geographical distribution: Union of Soviet Socialist Republics.

Induced disease: In winter wheat and rye, chlorotic mottling; profuse branching. In winter wheat, phloem necrosis; chloroplasts few, small; vacuolate inclusions in cells; nuclei enlarged and with extra nucleoli; no protein crystals of the pupation-disease type in affected cells. In spring wheat, barley, and oats, chlorotic mottling without profuse branching; no proliferation of flowers, but grain is rarely formed, most infected plants dying before this stage of growth.

Transmission: By leafhopper, *Deltocephalus striatus* L. (*CICADELLIDAE*), with incubation period of 15 to 18 days. Not by inoculation of expressed juice. Not through soil.

Literature: Zazhurilo and Sitnikova, Compt. rend. Acad. Sci. U. R. S. S., *25*, 1939, 798–801; *26*, 1940, 474–478; *29*, 1940, 429–432; Proc. Lenin Acad. Agr. Sci., U. R. S. S., *6*, 1941, 27–29. [Rev. Appl. Myc., *19*, 1940, 268; *20*, 1941, 157, 396; *22*, 1943, 59].

4. **Fractilinea quarta** (Holmes) *comb. nov.* (*Marmor quartum* Holmes, *loc. cit.*, 65.) From Latin *quartus*, fourth.

Common name: Sugar-cane chlorotic-streak virus or fourth-disease virus.

Host: *GRAMINEAE—Saccharum officinarum* L., sugar cane.

Geographical distribution: Java, Queensland, Hawaii, Puerto Rico, Colombia, United States (Louisiana).

Induced disease: In sugar cane, reduction of growth rate; wilting at midday; long, narrow, longitudinal streaks, of creamy or white color, in the leaves. Streaks 1/16 to 3/16 inches wide, generally less than 1 foot long, fragmenting.

Transmission: By leafhopper, *Draeculacephala portola* Ball (*CICADELLIDAE*). Not demonstrated by inoculation of expressed juice.

Thermal inactivation: In cuttings, at 52° C in less than 20 minutes.

Literature: Abbott, Phytopath., *28*, 1938, 855–857; Sugar Bull., *16*, 1938, 3–4; Abbott and Ingram, Phytopath., *32*, 1942, 99–100; Bell, Queensland Agr. Jour., *40*, 1933, 460–464; Martin, Hawaiian Planters' Rec., *34*, 1930, 375–378; Hawaiian Sugar Planters' Assoc. Proc., *53*, 1934, 24–35.

5. **Fractilinea zeae** (Holmes) *comb. nov.* (*Marmor zeae* Holmes, *loc. cit.*, 59.) From New Latin *Zea*, generic name for corn (maize), from Latin *zea*, a kind of grain.

Common name: Maize-stripe virus.

Host: *GRAMINEAE—Zea mays* L., corn (maize).

Insusceptible species: *GRAMINEAE —Saccharum officinarum* L., sugar cane.

Geographical distribution: Hawaii, Tanganyika, Mauritius, Trinidad, Cuba. Not in United States.

Induced disease: In corn (maize), at first few, elongated, chlorotic lesions near base of young leaf, later enlarging and fusing to form continuous stripes. Subsequently formed leaves banded and striped variously. Vacuolate intracellular inclusions in cells of affected areas.

Transmission: By leafhopper, *Peregrinus maidis* (Ashm.) (*FULGORIDAE*); the incubation period in this in-

sect is usually between 11 and 29 days, although shorter periods have been demonstrated in a few cases. Virus may persist in the insect host until death, but may become exhausted earlier. Not by aphid, *Aphis maidis* Fitch (*APHIDIDAE*). Not by inoculation of expressed juice.

Literature: Briton-Jones, Trop. Agr., *10*, 1933, 119–122; Carter, Ann. Ent. Soc. Am., *34*, 1941, 551–556; Kunkel, Bull. Hawaiian Sugar Planters' Assoc., Bot. Ser., *3*, 1921, 44–57; 1924, 108–114; Hawaiian Planters' Rec., *26*, 1922, 58–64; Phytopath., *17*, 1927, 41 (Abst.); Stahl, Trop. Pl. Res. Found., Bull. 7, 1927; Storey, Rept. of Plant Pathologist, Amani Agr. Res. Station, 4th Ann. Rept., 1931–32, pp. 8–13.

6. **Fractilinea avenae** McKinney. (Jour. Washington Acad. Sci., *34*, 1944, 327.) From Latin *avena*, oats.

Common name: Pupation-disease virus.

Hosts: *GRAMINEAE—Avena sativa* L., oat; *Triticum aestivum* L., wheat; *Echinochloa crusgalli* Beauv.; *Setaria viridis*; rarely, *Agropyron repens* (L.) Beauv. and *Bromus inermis* Leyss. Experimentally, also *Hordeum vulgare* L., barley; *Panicum miliaceum* L., millet; *Oryza sativa* L., rice; *Secale cereale* L., rye; *Zea mays* L., corn (maize).

Geographical distribution: West Siberia.

Induced disease: In oat, chlorotic mottling, profuse development of shoots, proliferation of flowers with change to leaf-like structures. Protein crystals in affected cells have been regarded as accumulated virus.

Transmission: By leafhopper, *Delphax striatella* Fallan (*FULGORIDAE*), especially first and second instar nymphs; fifth instar nearly immune to infection. Incubation period, 6 days or more. Virus overwinters in insect as well as in plants. Not transmitted from an infected leafhopper to its progeny. Not through soil. Not through seeds from infected plants.

Literature: Sukhov et al., Compt. rend. Acad. Sci., U. R. S. S., *20*, 1938, 745–748; *26*, 1940, 479–482, 483–486.

FAMILY II. MARMORACEAE HOLMES EMEND.

(Handb. Phytopath. Viruses, 1939, 16.)

Viruses of the Mosaic Group, inducing diseases usually characterized by persistent chlorotic or necrotic spotting, and often by mottling. The family is here extended to include several small groups of viruses, formerly assigned independent family rank, but sharing a tendency to aphid transmission, so far as known, and inducing diseases characterized by abnormal growth habit, thickening and rolling of leaves, or dwarfing, traits not incompatible with the characters of the present group. Should any one of these small groups become the center of a large assemblage of new viruses in the future, separate familial status for it might again be advantageous. In the combined grouping here used, specific vectors, so far as known, are aphids (*APHIDIDAE*).

Key to the genera of family **Marmoraceae.**

I. Viruses of the Typical Mosaic-Disease Group.
Genus I. *Marmor*, p. 1163.
II. Viruses of the Spindle-Tuber Group.
Genus II. *Acrogenus*, p. 1202.
III. Viruses of the Leaf-Roll Group.
Genus III. *Corium*, p. 1203.
IV. Viruses of the Dwarf-Disease Group.
Genus IV. *Nanus*, p. 1206.
V. Viruses of the Rough-Bark Group.
Genus V. *Rimocortius*, p. 1208.
VI. Viruses of the Symptomless Group.
Genus VI. *Adelonosus*, p. 1211.

Genus I. Marmor *Holmes.*

(*Loc. cit.*, 16)

Viruses inducing typical mosaic diseases in various plants. Generic name from Latin *marmor*, a mottled substance.

The type species is *Marmor tabaci* Holmes.

Key to the groups within genus **Marmor.**

A. Relatively resistant to heat inactivation, usually requiring more than 10 minutes at 85 to 90° C for complete inactivation.
1. Tobacco-Mosaic Virus Group.
B. Relatively susceptible to heat inactivation, requiring less than 10 minutes at 85 to 90° C for complete inactivation.
a. Replacing potato-veinbanding virus in mixed infections.
2. Tobacco-Etch Virus Group.
aa. Not replacing potato-veinbanding virus in mixed infections.
3. Cucumber-Mosaic Virus Group.
C. Many additional species cannot yet be grouped into definite subdivisions of the genus; they constitute a residual or
4. Miscellaneous Mosaic-Virus Group.

Key to the species of the Tobacco-Mosaic Virus Group.

Viruses relatively resistant to heat inactivation, requiring in most cases more than 10 minutes at 85 to 90° C for complete inactivation. Insect vectors as yet unknown under natural conditions.

I. Found in nature principally in solanaceous plants; *Cucurbitaceae* insusceptible. Chlorotic mottling in some hosts, necrotic lesions in others as result of experimental infection. Suspensions show anisotropy of flow.
1. *Marmor tabaci.*
2. *Marmor constans.*

II. Found in nature only in cucurbitaceous plants; *Solanaceae* insusceptible. Only mottling as result of experimental infection. Suspensions show marked anisotropy of flow.
3. *Marmor astrictum.*

III. Found only in leguminous plants. Chlorotic lesions in some varieties of the common snap-bean plant, necrotic lesions in others, as a result of experimental infection.
4. *Marmor lacsiofaciens.*

IV. Found in greenhouses confined to roots and lower parts of plants. Only necrotic lesions as result of experimental infection. Suspensions do not show anisotropy of flow.
5. *Marmor lethale.*

V. Found in tomato and experimentally transmissible to a number of species of plants in this and other families. Resembling the preceding in a number of physical characteristics, including failure to show anisotropy of flow.
5a. *Marmor dodecahedron.*

1. **Marmor tabaci** Holmes. (Holmes, Handb. Phytopath. Viruses, 1939, 17; *Musivum tabaci* Valleau, Phytopath., *30*, 1940, 822; *Phytovirus nicomosaicum* Thornberry, Phytopath., *31*, 1941, 23.) From New Latin *Tabacum*, early generic name for tobacco.

Common names: Tobacco-mosaic virus, tomato-mosaic virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., tobacco; *Lycopersicon esculentum* Mill., tomato; and *Capsicum frutescens* L., garden pepper, among crop plants; nearly all, if not all, solanaceous plants can be infected, although in some the virus remains localized at or near the site of inoculation. *PLANTAGINACEAE*—A strain of this virus has been found in nature infecting *Plantago lanceolata* L., ribgrass, *P. major* L. and *P. rugelii* Dcne., common broad-leaved plantains. Experimental hosts are widely distributed through many related families of plants.

Geographical distribution: World-wide.

Induced disease: In most varieties of tobacco, yellowish-green primary lesions, followed by clearing of veins, distortion and greenish-yellow mottling of newly formed leaves. In Ambalema tobacco, no symptoms, virus being restricted to inoculated leaves or those nearby. Strains of tobacco showing necrotic effects have been produced recently. In tomato, no obvious primary lesions, systemic disease characterized by greenish-yellow mottling of foliage, moderate distortion of leaf shape, and a reduction of fruit yield not exceeding 50 per cent. If some strain of potato-mottle virus (*Marmor dubium*) is also present, a more severe disease is induced; this is known as double-virus streak, and is characterized by systemic necrosis. In most varieties of garden pepper, yellowish primary lesions followed by systemic chlorotic mottling. In the Tabasco pepper and its recent derivatives, recovery by abscission of inoculated leaf, after localization of virus in necrotic primary lesions. Vacuolate intracellular inclusions are found in chlorotic tissues of all hosts that show distinct chlorotic mottling.

Transmission: By slight abrasive contacts. By inoculation of expressed juice. To some extent by the aphids, *Myzus pseudosolani* Theob., *M. circumflexus* (Buckt.), *Macrosiphum solanifolii* Ashm., and *Myzus persicae* (Sulz.) (*APHIDIDAE*). By grafting. Through soil.

Through dodder, *Cuscuta campestris* Yuncker (*CONVOLVULACEAE*), without infecting this plant vector. Not through pollen from diseased plants. Not through seeds from diseased tobacco; seed transmission has been reported in the case of recently ripened seeds from diseased tomato.

Serological relationships: Precipitin test gives cross reactions between all known strains, except those characterized by failure to spread systemically in tobacco. No cross reactions with other viruses except weakly with cucurbit-mosaic virus (*Marmor astrictum*). Type and other strains of tobacco-mosaic virus give cross reactions in complement-fixation and neutralization tests.

Immunological relationships: Plant protection tests, particularly in *Nicotiana sylvestris* Spegaz. and Comes, have demonstrated that tissues invaded by any strain of this virus are immune to subsequent infection by the tomato aucuba-mosaic strain of tobacco-mosaic virus, indicating a group relationship not shared by other viruses, such as cucumber-mosaic virus or tobacco-ringspot virus.

Thermal inactivation: At 88 to 93° C in 10 minutes; at 86 to 92° C in 30 minutes.

Filterability: Tobacco-mosaic virus was the first virus shown to be filterable, by Iwanowski in 1892; its filterability was confirmed and interpreted by Beijerinck in 1898.

Other properties: The ultimate particles of tobacco-mosaic virus have been shown to be rod-shaped and isotropic, sometimes associated in pairs, end to end. Under proper conditions, thread-like paracrystals are formed. Specific gravity has been determined as about 1.37, refractive index as about 1.6. Isoelectric point between pH 3.2 and 3.5. Suspensions in media of lower refractive indices show anisotropy of flow. Sedimentation constants, at 20° C, 187 $\times$ 10^{-13} cm per sec. per dyne at infinite dilution for unaggregated virus and 216 $\times$ 10^{-13} cm per sec. per dyne for associated particles. The computed average length of the virus unit is about 272 millimicrons; diameter, 13.8 millimicrons. Electron micrographs show that characteristic particles are rod-like, between 10 and 20 millimicrons in width, variable in length, but in some preparations averaging 270 millimicrons in length for single units, 405 to 540 millimicrons in length for associated pairs; X-ray measurements in air-dry gel show width 15.20 $\pm$ 0.05 millimicrons. Solutions stronger than about 1.3 per cent separate into layers, the lower spontaneously doubly refracting and more concentrated than the upper. At concentrations of electrolytes somewhat less than are required to precipitate the virus as fibres or needle-shaped paracrystals, the solutions form clear gels that become fluid on shaking or diluting (at pH 7 and 30° C). The virus is destroyed by high-frequency sound radiation, by pressures between 6000 and 8000 kilograms per square centimeter, and by hydrogen-ion concentrations above pH 11 or below pH 1. It is relatively stable between pH 2 and pH 8. It is rapidly broken down in 6 M urea solutions, in the presence of salts, to low-molecular-weight components devoid of activity. Analysis of purified virus: carbon 47.7 per cent, hydrogen 7.35 per cent, nitrogen 15.9 per cent, sulfur 0.24 per cent, phosphorus 0.60 per cent, lipoid 0.0 per cent, carbohydrate 1.6 to 2.0 per cent. A revised estimate of the sulfur content is 0.20 per cent, probably all in cysteine; no methionine has been detected in the typical variety of this virus. The percentages of the following substances in the virus are: tyrosine 3.9, tryptophane 4.5, proline 4.6, arginine 9.0, phenylalanine 6.0, serine 6.4, threonine 5.3, cysteine 0.68, alanine 2.4, aspartic acid 2.6, glutamic acid 5.3, leucine 6.1, valine 3.9, nucleic acid 5.8, and amide nitrogen 1.9, collectively accounting for about 68 per cent of the total weight. Virus formation ceases in infected host tissues immersed in 0.0002 molar sodium cyanide solution, beginning again after removal of cyanide.

Literature: The literature dealing with tobacco-mosaic virus is too voluminous to permit citation of more than a few representative publications. Allard, U. S. Dept. Agr., Bull. 40, 1914; Bawden and Pirie, Proc. Roy. Soc. London, Ser. B., *123*, 1937, 274–320; Beale, Jour. Exp. Med., *54*, 1931, 463–473; Beijerinck, Verhandel. Konink. Akad. Wetenschappen te Amsterdam, II, *6*, 1898, 3–22; Grant, Phytopath., *24*, 1934, 311–336; Hoggan, Jour. Agr. Res., *49*, 1934, 1135–1142; Iwanowski, Bull. Acad. Imp. Sci. St. Petersburg, Ser. 4, *5*, 1892, 67–70; Jensen, Phytopath., 23, 1933, 964–974; Johnson, Science, 64, 1926, 210; Kausche et al., Naturwiss., *27*, 1939, 292–299; Knight, Jour. Biol. Chem., *147*, 1943, 663–666; Kunkel, Phytopath., *24*, 1934, 437–466; Lauffer, Jour. Am. Chem. Soc., *66*, 1944, 1188–1194; Price, Phytopath., *23*, 1933, 749–769; Stanley, Phytopath., *26*, 1936, 305–320; Takahashi and Rawlins, Proc. Soc. Exp. Biol. and Med., *30*, 1932, 155–157; Valleau and Johnson, Kentucky Agr. Exp. Sta., Bull. 376, 1937; Vinson, Science, *66*, 1927, 357–358; Woods, Science, *91*, 1940, 295–296.

Strains: A great number of variant strains have been isolated both experimentally and from plants infected in nature. These usually share with the type variety most of the fundamental properties, particle size, especially width, stability at relatively high temperatures, longevity in storage, some common antigens. The following have been distinguished from the type, var. *vulgare* H. (*loc. cit.*, 17), by varietal names:

1a. *Marmor tabaci* var. *aucuba* H. (*loc. cit.*, 20). A group of isolates producing necrotic local lesions in inoculated leaves of *Nicotiana sylvestris* Spegaz. and Comes; useful in identifying many other strains of this virus which on prior application protect the tissues of this plant from the necrotic effects of aucuba-type strains (Smith, Ann. Appl. Biol., *18*, 1931, 471–493; Kunkel, Phytopath., *24*, 1934, 437–466).

1b. *Marmor tabaci* var. *deformans* H. (*loc. cit.*, 22). Producing exceptionally severe malformation of tomato foliage. (Ainsworth, Ann. Appl. Biol., *24*, 1937, 545–556).

1c. *Marmor tabaci* var. *canadense* H. (*loc. cit.*, 23). Producing a necrotic type of streak disease in tomatoes (Jarrett, Ann. Appl. Biol., *17*, 1930, 248–259).

1d. *Marmor tabaci* var. *lethale* H. (*loc. cit.*, 24). Producing spreading necrotic lesions in tobacco and tomato under experimental conditions (Jensen, Phytopath., *27*, 1937, 69–84; Norval, Phytopath. *28*, 1938, 675–692).

1e. *Marmor tabaci* var. *plantaginis* H. (Phytopath., *31*, 1941, 1097). Specially adapted in nature for systemic spread in species of *Plantago*. This variety contains histidine (0.55 per cent) and methionine (2 per cent) not found in the type of the species.

1f. *Marmor tabaci* var. *obscurum* H. (Handb. Phytopath. Viruses, 1939, 25). Systemic in tobacco without producing obvious disease under experimental conditions (Holmes, Phytopath., *24*, 1934, 845–873; *26*, 1936, 896–904; Jensen, Phytopath., *27*, 1937, 69–84).

1g. *Marmor tabaci* var. *immobile* H. (*loc. cit.*, 26). Produces chlorotic primary lesions in experimentally infected tobacco, but rarely becomes systemic. (Jensen, Phytopath., *23*, 1933, 964–977; *27*, 1937, 69–84).

1h. *Marmor tabaci* var. *artum* H. (*loc. cit.*, 27). Necrotic lesions experimentally induced in *Nicotiana glutinosa* L. (*SOLANACEAE*) are much smaller than those of the type variety (Jensen, Phytopath., *27*, 1937, 69–84).

1i. *Marmor tabaci* var. *siccans* Doolittle and Beecher. (Phytopath., *32*, 1942, 991). Causing necrosis and shriveling of tomato foliage.

2. **Marmor constans** McKinney. (Jour. Washington Acad. Sci., *34*, 1944, 326.) From Latin *constans*, fixed.

Common name: Tobacco mild dark-green mosaic virus.

Hosts: *SOLANACEAE—Nicotiana glauca* R. Grah., tree tobacco.

Insusceptible species: *SOLANACEAE—Lycopersicon esculentum* Mill., tomato. *CUCURBITACEAE—Cucumis sativus* L., cucumber.

Geographical distribution: Islands of Grand Canary and Teneriffe.

Induced disease: In *Nicotiana glauca*, systemic chlorotic mottling.

Transmission: By inoculation of expressed juice. No insect vector is known.

Thermal inactivation: At about 86°C. in 10 minutes.

Literature: McKinney, Jour. Agr. Res., *39*, 1929, 557–578; Am. Jour. Bot., *28*, 1941, 770–778; Peterson and McKinney, Phytopath., *28*, 1938, 329–342; Thornberry and McKinney, *ibid.*, *29*, 1939, 250–260.

3. **Marmor astrictum** Holmes. (Holmes, Handb. Phytopath. Viruses, 1939, 27; *Musivum astrictum* Valleau, Phytopath., *30*, 1940, 823.) From Latin *astrictus*, limited, in reference to host range.

Common names: Cucurbit-mosaic virus, English cucumber-mosaic virus.

Hosts: *CUCURBITACEAE—Cucumis sativus* L., cucumber; *C. anguria* L., gherkin; *C. melo* L., melon; *Citrullus vulgaris* Schrad., watermelon; only cucurbitaceous plants have appeared to be susceptible thus far.

Insusceptible species: All tested solanaceous species. *CUCURBITACEAE—Bryonia dioica* L.; *Cucurbita pepo* L., vegetable marrow. *LEGUMINOSAE—Phaseolus vulgaris* L. var. Golden Cluster.

Geographical distribution: England.

Induced disease: In cucumber, clearing of veins and crumpling in young leaves, followed by a green-mosaic mottling, with blistering and distortion of newly formed leaves. Plant stunted. Fruit unmarked or slightly mottled. Diseased plants less obviously affected during winter months.

Transmission: By inoculation of expressed juice. No insect vector is known.

Serological relationships: Weak cross-precipitin reactions and full cross-neutralization reactions with tobacco-mosaic virus (*Marmor tabaci*). Two common antigens postulated. Preparations of virus that have been inactivated by treatment with nitrous acid or X-rays are still antigenic.

Thermal inactivation: At 80 to 90° C in 10 minutes.

Filterability: Passes Pasteur-Chamberland filters L_1 to L_7, and membranes of 150 millimicrons average pore diameter.

Other properties: Virus, infectious in dilution of 10^{-10}, is present to the extent of 0.2 to 0.3 gram per liter of juice from diseased plants. Preparations show sheen and anisotropy of flow, indicating rod-shaped particles. Solutions stronger than 2.5 per cent separate into 2 layers at room temperature, the lower being the more concentrated and birefringent. Precipitates with ammonium sulfate show needle-shaped paracrystals. Sedimentation constants $S_{20}° = 173 \times 10^{-13}$ cm. sec.$^{-1}$ dyne^{-1} and about 200×10^{-13} cm. sec.$^{-1}$ dyne^{-1}. Virus withstands drying without inactivation but with partial loss of ability to show anisotropy of flow and with reduction of serological activity to about half. Tryptophane content 1.4 per cent, phenylalanine 10.2 per cent, the first lower and the second higher than in tobacco-mosaic virus.

Literature: Ainsworth, Ann. Appl. Biol., *22*, 1935, 55–67; Bawden and Pirie, Nature, *139*, 1937, 546–547; Brit. Jour. Exp. Path., *18*, 1937, 275–291; Knight, Arch. Virusf., *2*, 1942, 260–267; Knight and Stanley, Jour. Biol. Chem., *141*, 1941, 29–38; *141*, 1941, 39–49; Price, Am. Jour. Bot., *27*, 1940, 530–541; Price and Wyckoff, Nature, *141*, 1938, 685.

Strains: A distinctive strain has been distinguished from the type, var. *chlorogenus* H. (*loc. cit.*, 27), by the varietal name:

3a. *Marmor astrictum* var. *aucuba* H. (*loc. cit.*, 29). Differing from the type of this species by inducing a yellow-mottling, rather than a green-mottling, mosaic in cucumber (Ainsworth, Ann. Appl. Biol., *22*, 1935, 55–67).

4. **Marmor laesiofaciens** Zaumeyer and Harter. (Jour. Agr. Res., *67*, 1943, 305.) From Latin *laesio*, substantive from *laedere*, to injure, and participle from *facere*, to make.

Common name: Bean-mosaic virus 4; southern bean mosaic virus 1.

Hosts: *LEGUMINOSAE—Phaseolus vulgaris* L., bean. Experimentally, also *Phaseolus lunatus* L., sieva bean; *Soja max* Piper var. Virginia, Virginia soy bean.

Insusceptible species: All tested species in families other than the *LEGUMINOSAE*.

Geographical distribution: United States (Louisiana).

Induced disease: In bean, systemic chlorotic mottling in some varieties, localized necrosis in others; in a few varieties, systemic necrosis. In mottling-type varieties, chlorotic mottling of foliage; pods marked by dark green blotches or shiny areas, slightly malformed, short, frequently curled at end. In necrotic-type varieties with localized response, bearing a dominant gene lacking in mottling-type varieties, reddish necrotic lesions at the site of inoculation; no evidence of systemic spread of virus. In varieties showing systemic necrosis, pinpoint or slightly larger necrotic primary lesions with veinal necrosis of inoculated leaf; systemic veinal necrosis, distortion and curling of affected leaves, drooping at the pulvini; stem and petiole streak; eventual death of plant.

Transmission: By inoculation of expressed juice. Through seeds from infected plants.

Serological relationships: Not demonstrated.

Immunological relationships: Previous infection with bean-mosaic virus, *Marmor phaseoli*, does not protect against infection with this virus.

Thermal inactivation: At 90 to 95° C, time not stated, probably 10 minutes.

Other properties: Withstands dilution of 1:500,000 and aging 32 weeks at 18° C.

Literature: Zaumeyer and Harter, Phytopath., *32*, 1942, 438–439; *33*, 1943, 16; *34*, 1944, 510–512; Jour. Agr. Res., *67*, 1943, 295–300, 305–328.

Strains: A strain differing from the type has been given the varietal name:

4a. *Marmor laesiofaciens* var. *minus* Zaumeyer and Harter. (Jour. Agr. Res., *67*, 1943, 305.) From Latin *minor*, lesser. Differing from the type by inducing formation of slightly less diffuse and spreading lesions in necrotic-type bean leaves; also by inducing milder early symptoms and more severe late symptoms in mottling-type beans. Passes through seeds from infected plants to infect seedlings grown from them. Found in additional localities in the United States (California, Colorado, Idaho, Maryland).

5. **Marmor lethale** H. (*loc. cit.*, 86). From Latin *lethalis*, causing death.

Common name: Tobacco-necrosis virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., tobacco; *N. glutinosa* L.; *N. langsdorffii* Weinm.; *Lycopersicon esculentum* Mill., tomato; *Solanum nigrum* L. *COMPOSITAE*—Aster. *GERANIACEAE—Pelargonium hortorum* Bailey. *LEGUMINOSAE—Phaseolus vulgaris* L., bean. Confined to roots of these natural hosts except in the cases of *Nicotiana tabacum* and *N. glutinosa* in which lower leaves are sometimes invaded; necrotic lesions along midrib and larger veins in these. No obvious manifestations of disease in infected roots. Experimentally to plants in many families with production of localized necrotic lesions only.

Geographical distribution: England, Scotland, Australia. This virus has been found only in greenhouses.

Induced disease: In tobacco, necrosis of midrib and larger veins of first-developed pair of leaves, between November and February. Virus also in roots of many healthy-looking plants throughout the year. Upon artificial inoculation of foliage, numerous small brown necrotic local lesions are produced. Yield of virus from infected plant 0.02 mg per cc of expressed juice, on the average.

Transmission: By contamination of soil with virus. No insect vector is known. Experimentally, by inoculation of expressed juice.

Serological reactions: Precipitates with homologous antiserum. No cross reaction with tomato bushy-stunt or tobacco-mosaic viruses.

Immunological relationships: Protection tests show lack of relationship to tobacco-mosaic virus, tobacco-ringspot virus, tomato-ringspot virus, cucumber-mosaic virus, and the severe-etch strain of tobacco-etch virus.

Thermal inactivation: At 90 to 92° C in 10 minutes.

Filterability: Average particle diameter 20 to 30 millimicrons as determined by filtration through Gradocol membranes; other reports give diameter as 13 to 20 millimicrons by filtration (14 to 19 millimicrons by radiation experiments, about 20 millimicrons from electron micrographs).

Other properties: Infectious after storage for months in dried leaves and after storage for half a year in absolute ethyl alcohol at room temperature. Specific gravity 1.3. More soluble in ammonium sulfate solutions at 0° C than at room temperature. Composition: Carbon 44.8 to 45.3 per cent, nitrogen 15.5 to 16.5 per cent, hydrogen 6.5 to 7.0 per cent, phosphorus 1.4 to 1.7 per cent, sulfur 1.1 to 2.0 per cent, carbohydrate 7.0 to 9.0 per cent; ash 5.8 to 7.0 per cent (3 to 5 per cent after prolonged dialysis at pH 3). Nucleic acid of the ribose type has been isolated. No anisotropy of flow in solution but crystals are birefringent, showing sharp extinctions parallel to, and at right angles to, the plane of the crystal when examined edge-on in a polarizing microscope. Sedimentation constant, $S_{20}° = 112 \times 10^{-13}$; in other preparations a crystalline component with sedimentation constant 130×10^{-13} and an amorphous component with sedimentation constant 58×10^{-13} have been reported, as well as small amounts of a substance with sedimentation constant 220×10^{-13}.

Strains: Isolates of tobacco-necrosis virus serologically distinct but not otherwise different from each other appear to imply the existence of several strains of this virus, or of a closely related group of viruses, in England.

Literature: Bawden, Brit. Jour. Exp. Path., *22*, 1941, 59–70; Bawden et al., *ibid.*, *23*, 1942, 314–328; Cohen, Proc. Soc. Exp. Biol. and Med., *48*, 1941, 163–167; Lea, Nature, *146*, 1940, 137–138; Pirie et al., Parasitol., *30*, 1938, 543–551; Price, Am. Jour. Bot., *25*, 1938, 603–612; Am. Jour. Bot., *27*, 1940, 530–541; Arch. Virusf., *1*, 1940, 373–386; Price and Wyckoff, Phytopath., *29*, 1939, 83–94; Smith, Parasitol., *29*, 1937, 70–85; *29*, 1937, 86–95; Smith and Bald, Parasitol., *27*, 1935, 231–245; Smith and MacClement, Parasitol., *32*, 1940, 320–332.

5a. **Marmor dodecahedron** H. (*loc. cit.*, 30). From Greek *dōdekahedron*, dodecahedron.

Common name: Tomato bushy-stunt virus.

Hosts: *SOLANACEAE—Lycopersicon esculentum* Mill., tomato. Experimentally, also *SOLANACEAE—Datura stramonium* L.; *Nicotiana glutinosa* L.; *N. langsdorffii* Weinm.; *N. tabacum* L., tobacco; *Solanum nigrum* L. *LEGUMINOSAE—Phaseolus vulgaris* L., bean; *Vigna sinensis* (L.) Endl., cowpea. *COMPOSITAE—Zinnia elegans* Jacq., zinnia.

Geographical distribution: British Isles.

Induced disease: In tomato, some primary lesions necrotic, ring-like or spot-like, others masked, or disclosed only by chlorophyll retention in yellowing leaves. In young plants, systemic necrotic lesions may cause death; in older plants, growth ceases, young leaves become pale yellow; growing points may die, inducing growth of axillary buds to produce a bushy top; older leaves become yellowed and show some purple coloration. In White Burley tobacco, local necrosis only, lesions small, red at first, then white. In cowpea, reddish necrotic primary lesions only.

Transmission: By inoculation of expressed juice. Through dodder, *Cuscuta campestris* Yuncker (*CONVOLVULACEAE*). Not through seeds of diseased plants. No insect vector is known.

Serological relationships: A specific antiserum, prepared by a single intravenous injection of rabbits with 2 mg of purified virus, gives granular, compact precipitates, serving for quantitative estimation of this virus, antiserum being used at dilutions of 1:200 or 1:800, virus at dilutions to 10^{-6}.

Immunological relationships: Will infect plants previously invaded by tobacco-mosaic virus, tomato spotted-wilt virus, tobacco-ringspot virus, and Bergerac-ringspot virus.

Filterability: Passes membranes down to 40 millimicrons average pore diameter.

Other properties: Virus crystallizes from solutions of ammonium sulfate as isotropic, rhombic dodecahedra, which shrink and swell reversibly on drying and rewetting; shrinkage reduces size to 80 per cent of the wet dimensions. In the presence of heparin, non-birefringent prisms, rather than dodecahedra, appear. $S_{20}° = 132 \times 10^{-13}$ cm. sec.$^{-1}$ dyne^{-1}. Particle approximately spherical, 27.4 millimicrons in diameter by X-ray measurements (average diameter by filtration data, 14 to 20 millimicrons). Solutions do not show anisotropy of flow. Inactivated by drying. Molecular weight 8,800,000. Density 1.353. Molecular weight may be as high as 24,000,000 in solution, but the density is then lower, 1.286. Analysis: carbon 47 to 50 per cent, nitrogen 15.8 to 16.4 per cent, phosphorus 1.3 to 1.5 per cent, ash 1.7 to 5 per cent, hydrogen 7.2 to 8.2 per cent, sulfur 0.4 to 0.8 per cent, carbohydrate 5 to 6 per cent.

Literature: Ainsworth, Jour. Ministry Agr., *43*, 1936, 266–269; Bawden and Pirie, Nature, *141*, 1938, 513; Brit. Jour. Exp. Path., *19*, 1938, 251–263; Bernal and Fankuchen, Jour. Gen. Physiol., *25*, 1941, 111–165; Bernal et al., Nature, *142*, 1938, 1075; Cohen, Jour. Biol. Chem. *144*, 1942, 353–362; Proc. Soc. Exp. Biol. and Med., *51*, 1942, 104–105; Lauffer, Jour. Phys. Chem., *44*, 1940, 1137–1146; Lauffer and Stanley, Jour. Biol. Chem., *135*, 1940, 463–472; Neurath and Cooper, Jour. Biol. Chem., *135*, 1940, 455–462; Smith, Nature, *135*, 1935, 908; Ann. Appl. Biol., *22*, 1935, 731–741; Jour. Roy. Hort. Soc., *60*, 1935, 448–451; Smith and MacClement, Parasitol., *33*, 1941, 320–330; Stanley, Jour. Biol. Chem., *135*, 1940, 437–454.

Key to the species of the Tobacco-Etch Virus Group.

Viruses relatively susceptible to heat inactivation (inactivated at 52 to 58° C in 10 minutes). A small, closely allied group, tending to replace or to be replaced by each other, if present in mixture in tobacco.

I. Not replaced, if in mixture, by other viruses of this group; dominant member of the group in tobacco.

6. *Marmor erodens.*

II. Replaced by No. 6, not by No. 8, if in mixture with it in tobacco.

7. *Marmor hyoscyami.*

III. Replaced by No. 6 or 7 if in mixture with either in tobacco.

8. *Marmor upsilon.*

6. **Marmor erodens** Holmes. (Holmes, Handb. Phytopath. Viruses, 1939, 40; *Foliopellis erodens* Valleau, Phytopath., *30*, 1940, 825.) From Latin *erodere*, to erode or gnaw away.

Common name: Tobacco-etch virus.

Hosts: *SOLANACEAE—Capsicum frutescens* L., pepper; *Datura stramonium* L., Jimson weed; *Lycopersicon esculentum* Mill., tomato; *Nicotiana tabacum* L., tobacco; *Petunia* sp., petunia; *Physalis heterophylla* Nees.

Geographical distribution: United States.

Induced disease: In tobacco, systemic mild-mottling chlorosis, with traces of necrotic etching; intranuclear crystalline inclusions and intracytoplasmic granular and amorphous inclusions that tend to crystallize, forming needle-shaped birefringent bodies, 2 to 10 microns in length.

Transmission: Experimentally, by *Myzus persicae* (Sulz.), *M. circumflexus* (Buckt.), *Aphis rhamni* Boyer, *A. fabae* (Scop.), and *Macrosiphum gei* (Koch) (*APHIDIDAE*); by inoculation of expressed juice.

Serological relationships: Precipitin reactions with homologous antisera, but no cross-reactions with tobacco-mosaic virus, tobacco-ringspot virus, potato-mottle virus, potato aucuba-mosaic virus, potato mild-mosaic virus, hyoscyamus-mosaic virus, potato-veinbanding virus, or pea-mosaic virus.

Immunological relationships: Protects tobacco against subsequent infection by potato-veinbanding virus and hyoscyamus-mosaic virus. In mixed infections, it suppresses and replaces these two viruses.

Thermal inactivation: At 53 to 55° C in 10 minutes.

Filterability: Passes Pasteur-Chamberland L_1, not L_3, filter candle.

Other properties: Sedimentation constant $S_{20}° = 170 \times 10^{-13}$ cm. sec.$^{-1}$ dyne^{-1}. Concentrated preparations show anisotropy of flow, indicating elongated particle shape.

Literature: Bawden and Kassanis, Ann. Appl. Biol., *28*, 1941, 107–118; Fernow, Cornell Agr. Exp. Sta. (Ithaca), Mem. 96, 1925; Holmes, Phytopath., *32*, 1942, 1058–1067; Johnson, Kentucky Agr. Exp. Sta., Res. Bull. 306, 1930.

Strains: A distinctive severe-symptom strain, isolated from plants infected in nature and studied intensively, has been distinguished from the type, var. *vulgare* H. (*loc. cit.*, 40), by the varietal name:

6a. *Marmor erodens* var. *severum* H. (*loc. cit.*, 41). Differing from the type by a tendency to induce more pronounced necrotic etching and a greater stunting effect in infected tobacco.

7. **Marmor hyoscyami** *spec. nov.* From New Latin *Hyoscyamus*, genus name of plant from which this virus was first isolated.

Common names: Hyoscyamus-mosaic virus, Hy. III virus, Hyoscyamus-III-disease virus.

Hosts: *SOLANACEAE—Hyoscyamus niger* L., henbane. Experimentally, also *Nicotiana tabacum* L., tobacco.

Insusceptible species: *CUCURBITACEAE—Cucumis sativus* L., cucumber.

Geographical distribution: England.

Induced disease: In henbane, chlorotic clearing of veins followed by yellow-mottling mosaic.

Transmission: By inoculation of expressed juice to dilutions of 10^{-4}. By aphids, *Myzus persicae* (Sulz.), *M. circumflexus* (Buckt.), and *Macrosiphum solanifolii* Ashm. (= *M. gei* Koch) (*APHIDIDAE*).

Serological relationships: Several isolates of this virus give mutual cross-precipitin reactions but no precipitation occurs when antiserum prepared with this virus is mixed with cucumber-mosaic virus, tobacco-etch virus, or potato-veinbanding virus.

Immunological relationships: No immunity with respect to this virus is induced in tobacco by previous infection with cucumber-mosaic virus. Potato-veinbanding virus is unable to multiply in the presence of this virus and is replaced by it. Tobacco-etch virus pro-

tects against this virus and replaces it in mixed infections.

Thermal inactivation: At 58° C in 10 minutes.

Filterability: Passes Chamberland L_1, but not L_2, filter candles.

Other properties: Concentrated solutions show anisotropy of flow. Yield of virus, 1 to 3 mg per liter of juice expressed from diseased tobacco plants.

Literature: Bawden and Kassanis, Ann. Appl. Biol., *28*, 1941, 107–118; Hamilton, *ibid.*, *19*, 1932, 550–567; Sheffield, *ibid.*, *25*, 1938, 781–789; Watson and Roberts, Proc. Roy. Soc. London, Ser. B, *127*, 1939, 543–576.

8. **Marmor upsilon** *comb. nov.* (*Marmor cucumeris* var. *upsilon* Holmes, *loc. cit.*, 33; *Murialba venataenia* Valleau, Phytopath., *30*, 1940, 824.) From Greek name of the letter Y, sometimes used to denote this virus.

Common names: Potato-veinbanding virus, potato virus Y.

Hosts: *SOLANACEAE—Solanum tuberosum* L., potato; *Nicotiana tabacum* L., tobacco. Experimentally, also *Lycium barbarum* L.

Geographical distribution: England, France, United States, Brazil. Rare in Scotland and part of Ireland.

Induced disease: In some potato varieties, leaf drop and necrotic stem-streak; in others, no signs of disease; in still others, chlorotic mottling with or without necrosis. In combination with strains of the potato-mottle virus (*Marmor dubium*), this virus causes rugose mosaic, a common and destructive double-virus disease.

Transmission: By inoculation of expressed juice. By aphid, *Myzus persicae* (Sulz.); experimentally, also by *Aphis rhamni* Boyer (synonym for *Aphis abbreviata* Patch) (*APHIDIDAE*).

Serological relationships: Precipitin reactions with homologous antisera. No cross reactions with tobacco-mosaic virus, tobacco-etch virus, hyoscyamus-mosaic virus, potato-mottle virus, potato mild-mosaic virus, potato aucuba-mosaic virus, tobacco-ringspot virus, or common pea-mosaic virus. Reported cross reaction with cucumber-mosaic virus needs confirmation.

Immunological relationships: A mild strain protects against subsequent infection with the typical virus. This virus is suppressed and replaced by hyoscyamus-mosaic virus and by tobacco-etch virus in mixed infections.

Thermal inactivation: At 52° C in 10 minutes.

Filterability: Passes with difficulty through Gradocol membrane of 42 millimicron average pore diameter.

Other properties: Inactivated by drying.

Literature: Dennis, Nature, *142*, 1938, 154; Johnson, Phytopath., *25*, 1935, 650–652; Jones and Vincent, Jour. Agr. Res., *55*, 1937, 69–79; Kassanis, Ann. Appl. Biol., *29*, 1942, 95; Koch, Phytopath., *23*, 1933, 319–342; Kramer and Silberschmidt, Arquivos Inst. Biol., São Paulo, Brazil, *11*, 1940, 165–188; Salaman, Nature, *159*, 1937, 924; Smith, Proc. Roy. Soc., Ser. B, *109*, 1931, 251–267; Smith and Dennis, Ann. Appl. Biol., *27*, 1940, 65–70.

Key to the species of the Cucumber-Mosaic Virus Group.

Viruses relatively susceptible to heat inactivation, requiring less than 10 minutes at 85 to 90° C for complete inactivation. Not replacing potato-veinbanding virus in mixed infections.

I. Infecting both dicotyledonous and monocotyledonous plants.
   9. *Marmor cucumeris.*

II. Infecting dicotyledonous, but not monocotyledonous, plants.
   10. *Marmor solani.*
   11. *Marmor aucuba.*
   12. *Marmor umbelliferarum.*

13. *Marmor cruciferarum.*
14. *Marmor brassicae.*
15. *Marmor betae.*
16. *Marmor lactucae.*
17. *Marmor dahliae.*
18. *Marmor phaseoli.*
19. *Marmor leguminosarum.*
20. *Marmor pisi.*
21. *Marmor medicaginis.*

III. Infecting monocotyledonous, but not dicotyledonous, plants.

22. *Marmor tulipae.*
23. *Marmor mite.*
24. *Marmor iridis.*
25. *Marmor sacchari.*
26. *Marmor cepae.*
27. *Marmor scillearum.*

9. **Marmor cucumeris** Holmes. (Holmes, Handb. Phytopath. Viruses, 1939, 31; *Murialba cucumeris* Valleau, Phytopath., *30*, 1940, 823.) From Latin *cucumis*, cucumber.

Common name: Cucumber-mosaic virus.

Hosts: Very wide range of hosts among dicotyledonous and monocotyledonous plants; cucumber, celery, spinach, tobacco, and pepper are sometimes seriously affected. Overwintering hosts are: *SOLANACEAE—Physalis subglabrata* Mackenzie and Bush, *P. heterophylla* Nees. *ASCLEPIADACEAE—Asclepias syriaca* L. *PHYTOLACCACEAE—Phytolacca decandra* L. *LABIATAE—Nepeta cataria* L. Probably there are also other susceptible perennials.

Geographical distribution: Probably almost world-wide.

Induced disease: In cucumber, *Cucumis sativus* L., yellowish-green systemic mottling. Leaves small, distorted, curled; plants dwarfed, internodes shortened. Few fruits set. Fruits mottled, misshapen, giving the disease the name "white pickle." In black cowpea, *Vigna sinensis* (L.) Endl., small reddish necrotic local lesions only. No intracellular bodies are found in plants infected with cucumber-mosaic virus.

Transmission: By inoculation of expressed juice. By aphids, *Myzus persicae* (Sulz.), *M. pseudosolani* Theob., *M. circumflexus* (Buckt.), *Macrosiphum solanifolii* Ashm., and *Aphis gossypii* Glov. (*APHIDIDAE*). Through seeds of diseased plants in *Echinocystis lobata* (Michx.) Torr. and Gray, wild cucumber, in *Cucumis melo* L., muskmelon, and in *Cucurbita pepo* L., vegetable marrow. By several species of dodder, *Cuscuta californica* Choisy, *C. campestris* Yuncker, and *C. subinclusa* Dur. and Hilg. (*CONVOLVULACEAE*).

Immunological relationships: Infection with the type and other chlorotic-mottling strains protects zinnia against subsequent infection by an indicator strain of this virus (var. *judicis*).

Thermal inactivation: At 70 to 80° C in 10 minutes.

Filterability: Passes Berkefeld W and N filters and collodion membranes of 45 millimicron average pore diameter.

Other properties: Inactivated by drying or 3 to 4 days' storage in juice at room temperature.

Literature: Ainsworth, Ann. Appl. Biol., *25*, 1938, 867–869; Chamberlain, New Zealand Jour. Science and Technology, *21*, 1939, 73A–90A; Celino, Philippine Agr., *29*, 1940, 379–414; Doolittle, Phytopath., *6*, 1916, 145–147; U. S. Dept. Agr., Bull. 879, 1920; Doolittle and Walker, Jour. Agr. Res., *31*, 1925, 1–58; Gilbert, Phytopath., *6*, 1916, 143–144; Hoggan, Jour. Agr. Res., *47*, 1933, 689–704; Jagger, Phytopath., *6*, 1916, 148–151;

8, 1918, 32–33; Kendrick, Phytopath., *24*, 1934, 820–823; Mahoney, Proc. Am. Soc. Hort. Sci., *332*, 1935, 477–480; Price, Phytopath., *25*, 1935, 776–789; *29*, 1939, 903–905; Am. Jour. Bot., *27*, 1940, 530–541; Storey, Ann. Appl. Biol., *26*, 1939, 298–308.

Strains: Various host plants seem to have induced specialization of cucumber-mosaic virus in strains particularly adapted to existence in their tissues. Several of these and certain laboratory-derived strains useful in technical procedures have been distinguished from the type, var. *vulgare* H. (*loc. cit.*, 31), by varietal names, as follows:

9a. *Marmor cucumeris* var. *commelinae* H. (*loc. cit.*, 35). From New Latin *Commelina*, generic name of weed serving as a natural reservoir of this strain. Common name: Southern celery-mosaic strain of cucumber-mosaic virus. Differing from the type in severity of disease induced in celery and some other plants. Transmitted by *Aphis gossypii* Glov., *A. maidis* Fitch, and *Pentalonia nigronervosa* Coq. (*APHIDIDAE*). (Price, Phytopath., *25*, 1935, 947–954; Wellman, *ibid.*, *24*, 1934, 695–725, 1032–1037; *25*, 1935, 289–308, 377–404.)

9b. *Marmor cucumeris* var. *phaseoli* H. (*loc. cit.*, 36). From New Latin *Phaseolus*, generic designation of lima bean. Common name: Lima-bean strain of cucumber-mosaic virus. Differing from type of species in ability to cause a chlorotic mottling disease in lima bean in nature. (Harter, Phytopath., *26*, 1936, 94; Jour. Agr. Res., *56*, 1938, 895–906; McClintock, Phytopath., *7*, 1917, 60.)

9c. *Marmor cucumeris* var. *lilii* H. (*loc. cit.*, 37). From Latin *lilium*, lily. Common name: Lily-mosaic strain of cucumber-mosaic virus. Differing from the type variety by ability to persist in nature in lilies, producing masked infection or chlorotic mottling unless in mixture with lily-symptomless virus (*Adelonosus lilii*), when a more severe disease involving necrosis is induced. (Brierley, Phytopath., *29*, 1939, 3; *30*, 1940, 250–257; Brierley and Doolittle, *ibid.*, *30*, 1940, 171–174; Ogilvie and Guterman, *ibid.*, *19*, 1929, 311–315; Price, *ibid.*, *27*, 1937, 561–569.)

9d. *Marmor cucumeris* var. *judicis* H. (*loc. cit.*, 38). From Latin *judex*, judge. Common name: Indicator strain of cucumber-mosaic virus. Differing from the type variety in inducing the formation of necrotic local lesions in zinnia (*Zinnia elegans* Jacq., *COMPOSITAE*). Previous infection of zinnia by other strains of cucumber-mosaic virus inhibits the formation of these necrotic local lesions, identifying the strains as related to each other and to the indicator strain. (Price, Phytopath., *24*, 1934, 743–761; *25*, 1935, 776–789.)

9e. *Marmor cucumeris* var. *vignae* H. (*loc. cit.*, 39). From New Latin *Vigna*, generic name of cowpea. Common name: Cowpea-mottling strain of cucumber-mosaic virus. Differing from the type variety in producing systemic chlorotic mottling, rather than reddish-brown necrotic local lesions, in Black cowpea. Not known in nature but derived experimentally from a mild-mottling strain of cucumber-mosaic virus during serial passage in cowpea. (Price, Phytopath., *24*, 1934, 743–761; *25*, 1935, 776–789.)

10. **Marmor solani** H. (*loc. cit.*, 47). From New Latin *Solanum*, generic name of potato.

Common names: Potato mild-mosaic virus, potato virus A.

Hosts: *SOLANACEAE—Solanum tuberosum* L., potato. Experimentally, also *Nicotiana tabacum* L., tobacco; *Solanum nigrum* L. var. *nodiflorum*; and *Datura stramonium* L., Jimson weed.

Geographical distribution: United States, England, Holland.

Induced disease: In potato, very mild chlorotic mottling or masked symptoms in some varieties (as Irish Chieftain), systemic necrosis in others (for example,

British Queen). Immunity to aphid infection with this virus is found in the varieties Katahdin and Earlaine. A combination disease, characterized by pronounced yellow-mosaic patterns, is caused by this virus in the variety Irish Chieftain if the potato-veinbanding virus (*Marmor upsilon*) is also present. In tobacco, experimentally, faint veinbanding mosaic.

Transmission: To potato, by rubbing methods of inoculation of expressed juice, using carborundum powder; to tobacco, by rubbing without carborundum. By aphids, *Aphis abbreviata* Patch and *Myzus persicae* (Sulz.) (*APHIDIDAE*).

Serological relationships: No cross-precipitin reactions with potato aucuba-mosaic virus, potato-veinbanding virus, tobacco-mosaic virus, tobacco-etch virus, tobacco-ringspot virus, or pea-mosaic virus.

Immunological relationships: A feeble strain of this virus has been found to protect fully against the typical strain in the Netherlands.

Thermal inactivation: At 50° C in 10 minutes.

Literature: Bawden, Ann. Appl. Biol., *23*, 1936, 487–497; Chester, Phytopath., *25*, 1935, 686–701; Dykstra, Phytopath., *29*, 1939, 40–67; Hansen, Tidsskr. Planteavl, *42*, 1937, 631–681; Murphy and Loughnane, Sci. Proc. Roy. Dublin Soc., *21*, 1936, 419–430; Murphy and McKay, *ibid.*, *20*, 1932, 227–247; Oortwijn Botjes, Tijdsch. Plantenziekten, *45*, 1939, 25–29; Schultz *et al.*, Phytopath., *27*, 1937, 190–197; *30*, 1940, 944–951.

11. **Marmor aucuba** H. (*loc. cit.*, 49). From New Latin *Aucuba*, a genus of plants having mottled foliage.

Common name: Potato aucuba-mosaic virus.

Hosts: *SOLANACEAE—Solanum tuberosum* L., potato. Experimentally, also *Atropa belladonna* L. (symptomless); *Capsicum frutescens* L., pepper; *Datura stramonium* L., Jimson weed (symptomless); *Hyoscyamus niger* L., henbane (symptomless); *Lycopersicon esculentum* Mill., tomato; *Petunia hybrida* Vilm., *petunia* (symptomless); *Nicotiana tabacum* L., tobacco (symptomless); *Solanum dulcamara* L., bittersweet; *S. nigrum* L. var. *nodiflorum*.

Geographical distribution: United States, Great Britain, Europe.

Induced disease: In potato, yellow spots on lower leaves of some varieties; in the variety Irish Chieftain, brilliant yellow mottle over whole plant, perhaps because of simultaneous presence of potato mild-mosaic virus in this variety. Necrosis of the cortex and of the pith in tubers in many varieties.

Transmission: By inoculation of expressed juice. Probably by aphid, *Myzus persicae* (Sulz.) (*APHIDIDAE*).

Serological relationships: No precipitin cross-reactions with potato mild-mosaic virus, potato-veinbanding virus, tobacco-mosaic virus, tobacco-etch virus, tobacco-ringspot virus, or pea-mosaic virus. Precipitin cross-reactions with the Canada-streak strain of potato aucuba-mosaic virus.

Thermal inactivation: At 65 to 68° C in 10 minutes.

Filterability: Passes Pasteur-Chamberland L_1 filter, but not L_3 or L_5.

Literature: Chester, Phytopath., *25*, 1935, 686–701; *27*, 1937, 903–912; Clinch, Sci. Proc. Roy. Dublin Soc., *22*, 1941, 435–445; Clinch et al., *ibid.*, *21*, 1936, 431–448; Dykstra, Phytopath., *29*, 1939, 917–933.

Strains: One strain differing from the type has been given a varietal name:

11a. *Marmor aucuba* var. *canadense* Black and Price. (Phytopath. *30*, 1940, 444.) From common name of strain.

Common name: Canada-streak strain of potato aucuba-mosaic virus. Differing from the type variety by tendency to produce necrosis in stems, veins, petioles, and leaves and also, about 2 months after harvest, in pith of tuber, especially at stem end. (Chester, Phytopath., *27*,

1937, 903–912; Dykstra, Phytopath., *29*, 1939, 917–933.)

12. **Marmor umbelliferarum** H. (*loc. cit.*, 67). From New Latin *Umbelliferae*, family name of plants among which celery is classified.

Common name: Celery-mosaic virus, western celery-mosaic virus.

Hosts: *UMBELLIFERAE—Apium graveolens* L., celery and celeriac; *Daucus carota* L., carrot. Experimentally, also *Anethum graveolens* L., dill; *Anthriscus cerefolium* (L.) Hoffm., salad chervil; *Carum carvi* L., caraway; *Coriandrum sativum* L., coriander; *Petroselinum hortense* Hoffm., parsley.

Insusceptible species: *Cucumis sativus* L., cucumber, and all other tested species not of the family *Umbelliferae*.

Geographical distribution: United States (California).

Induced disease: In celery, at first, clearing of veins in young leaves; later, foliage yellowed, plant stunted, young petioles shortened, older petioles horizontal, giving plant a flat appearance. Foliage mottled green and yellow; leaflets narrow, twisted or cupped; older leaves with some necrosis; petioles with white streaks or spots. In celeriac, clearing of veins, followed by systemic chlorotic mottling. In carrot, chlorotic spotting of young leaves, followed by systemic chlorotic mottling.

Transmission: By inoculation of expressed juice, in dilutions to 1:4000. No specific insect vector is known, but 11 species of aphids capable of breeding on celery transmit the virus, though they do not long retain the power of transmission after leaving diseased plants. These vectors are *Aphis apigraveolens* Essig, *A. apii* Theob., *A. ferruginea-striata* Essig, *A. gossypii* Glov., *A. middletonii* Thomas, *A. rumicis* Linn., *Cavariella capreae* (Fabr.), *Myzus circumflexus* (Buckt.), *M. convolvuli* (Kalt.), *M. persicae* (Sulz.), *Rhopalosiphum melliferum* (Hottes) (*APHIDIDAE*). Some aphids not able to breed on celery also transmit this virus.

Thermal inactivation: At 55 to 60° C in 10 minutes.

Filterability: Passes all grades of Chamberland filters.

Other properties: Virus active after storage at −18° C for 18 months.

Literature: Severin and Freitag, Hilgardia, *11*, 1938, 493–558.

13. **Marmor cruciferarum** H. (*loc. cit.*, 69). From New Latin *Cruciferae*, family name of plants among which cauliflower is classified.

Common name: Cauliflower-mosaic virus.

Hosts: *CRUCIFERAE—Brassica oleracea* L., cauliflower, kale, Brussels sprouts, cabbage, and broccoli; *B. campestris* L., wild yellow mustard; *Matthiola incana* R. Br., annual stock. Experimentally, also *Brassica adpressa* Boiss; *B. alba* Rabenh., white mustard; *B. arvensis* (L.) Ktze., charlock; *B. juncea* Coss., leaf mustard (one strain not susceptible); *B. napus* L., rape; *B. petsai* Bailey, pe-tsai; *B. nigra* Koch, black mustard; *B. rapa* L., turnip; *Capsella bursa-pastoris* Medic., shepherd's purse; *Iberis amara* L., rocket candytuft; *Lepidium sativum* L., garden cress; *Lunaria annua* L., honesty; *Raphanus raphanistrum* L., white charlock; *R. sativus* L., radish.

Insusceptible species: *CHENOPODIACEAE—Spinacia oleracea* L. *COMPOSITAE—Lactuca sativa* L. *CRUCIFERAE—Alyssum saxatile* L.; *A. maritimum* Lam.; *Arabis albida* Stev.; *Athysanus pusillus* Greene; *Brassica juncea* Coss. (Japanese strain; another strain susceptible); *Cheiranthus cheiri* L.; *Erysimum perofskianum* Fisch. and Mey.; *Hesperis matronalis* L.; *Malcomia maritima* R. Br.; *Roripa nasturtium* Rusby; *Stanleya pinnata* (Pursh.) Britt.; *Thysanocarpus radians* Benth. *LEGUMINOSAE—Vicia faba* L. *SOLANACEAE—Capsicum frutescens* L.; *Lycoper-*

sicon esculentum Mill.; *L. pimpinellifolium* Mill.; *Nicotiana glutinosa* L.; *N. langsdorffii* Weinm.; *N. tabacum* L. vars. Turkish and White Burley. *TROPAEOLACEAE—Tropaeolum majus* L. *UMBELLIFERAE—Apium graveolens* L.

Geographical distribution: United States, England.

Induced disease: In cauliflower, clearing of veins, followed by mild chlorotic mottling, veins usually banded with dark green, necrotic flecks later in chlorotic areas. Midrib curved, leaves distorted. Plant stunted; terminal head or curd dwarfed. Solanaceous plants appear to be immune, a point of distinction between this virus and turnip-mosaic virus, *Marmor brassicae*.

Transmission: By inoculation of expressed juice, using carborundum powder. By many aphid species, *Brevicoryne brassicae* (Linn.), cabbage aphid; *Rhopalosiphum pseudobrassicae* Davis, false cabbage aphid; *Myzus persicae* (Sulz.), peach aphid; *Aphis graveolens* Essig, celery leaf aphid; *A. apigraveolens* Essig, celery aphid; *A. middletonii* Thomas, erigeron root aphid; *A. gossypii* Glov., cotton aphid; *Cavariella capreae* (Fabr.), yellow willow aphid; *Myzus circumflexus* (Buckt.), lily aphid; *Rhopalosiphum melliferum* (Hottes), honeysuckle aphid. (*APHIDIDAE*). No seed transmission.

Thermal inactivation: At 75° C in 10 minutes.

Literature: Caldwell and Prentice, Ann. Appl. Biol., *29*, 1942, 366–373, 374–379; Rawlins and Tompkins, Phytopath., *24*, 1934, 1147 (Abst.); Tompkins, Jour. Agr. Res., *55*, 1937, 33–46.

14. Marmor brassicae H. (H., *loc. cit.*, 70; *Marmor matthiolae* H., *loc. cit.*, 71.) From New Latin, *Brassica*, generic name of turnip.

Common name: Turnip-mosaic virus.

Hosts: *CRUCIFERAE—Brassica rapa* L., turnip; *B. napobrassica* Mill., swede or rutabaga; *B. napus* L., rape; *B. nigra* (L.) Koch, black mustard; *B. oleracea* L., cabbage; *Armoracia rusticana* Gaertn., horse-radish; *Cheiranthus cheiri* L., wallflower; *Matthiola incana* R. Br., stock; *Sinapis alba* L., white mustard. Experimentally, also *CRUCIFERAE—Berteroa incana* (L.) DC.; *Brassica alba* Rabenh., white mustard; *B. arvensis* (L.) Ktze.; *B. chinensis* L., Chinese cabbage; *B. juncea* (L.) Coss.; *Capsella bursa-pastoris* (L.) Medic.; *Cardamine heterophylla* (Forst. f.) O. E. Schultz; *Cheiranthus allionii* Hort.; *Coronopus didymus* Smith; *Hesperis matronalis* L.; *Lepidium ruderale* L.; *L. sativum* L., *L. virginicum* L.; *Nasturtium officinale* R. Br.; *Neslia paniculata* (L.) Desv.; *Radicula palustris* (L.) Moench.; *Raphanus sativus* L.; *Sisymbrium altissimum* L.; *S. officinale* (L.) Scop.; *Thlaspi arvense* L. *CHENOPODIACEAE—Beta vulgaris* L.; *Spinacia oleracea* L., spinach. *COMPOSITAE—Calendula officinalis* L. *Zinnia elegans* Jacq. *RANUNCULACEAE—Delphinium ajacis* L. *SOLANACEAE—Lycopersicon pimpinellifolium* Mill.; *Nicotiana bigelovii* S. Wats.; *N. glutinosa* L.; *N. langsdorffii* Weinm.; *N. repanda* Willd.; *N. rustica* L.; *N. sylvestris* Speg. and Comes; *N. tabacum* L., tobacco; *Petunia hybrida* Vilm.

Geographical distribution: United States, England, New Zealand.

Induced disease: In turnip, systemic chlorotic mottling; plants stunted, leaves distorted. In tobacco, experimentally, characteristic necrotic primary lesions only.

Transmission: By inoculation of expressed juice. By cabbage aphid, *Brevicoryne brassicae* (Linn.), and by the peach aphid, *Myzus persicae* (Sulz.) (*APHIDIDAE*).

Thermal inactivation: At 54° C in 10 minutes.

Strains: A considerable number of strains of this virus appear to occur in nature, but those that have been studied often have been considered as distinct viruses and not compared with each other

under identical circumstances. More work is needed to show existing alliances.

Literature: Chamberlain, New Zealand Jour. Agr., *53*, 1936, 321–330; New Zealand Jour. Science and Technology, *21*, 1939, 212A–223A; Clayton, Jour. Agr. Res., *40*, 1930, 263–270; Gardner and Kendrick, *ibid.*, *22*, 1921, 123–124; Hoggan and Johnson, Phytopath., *25*, 1935, 640–644; Larson and Walker, Jour. Agr. Res., *59*, 1939, 367–392; *62*, 1941, 475–491; Schultz, Jour. Agr. Res., *22*, 1921, 173–178; Smith, Ann. Appl. Biol., *22*, 1935, 239–242; Tompkins, Jour. Agr. Res., *57*, 1938, 589–602; *58*, 1939, 63–77; Tompkins *et al.*, *ibid.*, *57*, 1938, 929–943.

15. **Marmor betae** H. (*loc. cit.*, 72). From Latin *beta*, beet.

Common name: Sugar-beet mosaic virus.

Hosts: *CHENOPODIACEAE—Beta vulgaris* L., beet; *Spinacia oleracea* L., spinach.

Geographical distribution: France, Denmark, Germany, Sweden, United States, England.

Induced disease: In beet, discrete yellowish secondary lesions or clearing of veins on young leaves, followed by chlorotic mottling of newly formed leaves. Darkening of vascular tissue. Leaves bend back near tips, which sometimes die. Intracellular bodies formed. In spinach, 6 to 21 days after infection, chlorotic flecks on young leaves. Plant stunted, outer leaves killed, dying from their tips back. Center of plant survives for a time, but finally dies.

Transmission: By inoculation of expressed juice, in dilutions to 10^{-3}. By aphids, *Myzus persicae* (Sulz.), *Aphis rumicis* Linn., and perhaps *Macrosiphum solanifolii* Ashm. (= *M. gei* Koch) (*APHIDIDAE*). No seed transmission.

Thermal inactivation: At 55 to 60° C in 10 minutes.

Other properties: Inactivated by standing in expressed juice for 24 to 48 hours at about 70° F.

Literature: Böning, Forsch. Geb. Pflanzenkr. u. Immun. Pflanzenreich, *3*, 1927 81–128; Cent. f. Bakt., II Abt., *71*, 1927, 490–497; Gratia and Manil, Compt. rend. Soc. Biol., Paris, *118*, 1935, 379–381; Hoggan, Phytopath., *23*, 1933, 446–474; Jones, Washington Agr. Exp. Sta. Bull. 250, 1931; Lind, Tidsskr. Planteavl, *22*, 1915, 444–457; Robbins, Phytopath., *11*, 1921, 349–365; Schmidt, Ber. Deutsch. Bot. Ges., *45*, 1927, 598–601.

16. **Marmor lactucae** H. (*loc. cit.*, 84). From Latin *lactuca*, lettuce.

Common name: Lettuce-mosaic virus.

Hosts: *COMPOSITAE—Lactuca sativa* L., lettuce; *Senecio vulgaris* L., groundsel. Experimentally, also *COMPOSITAE—Sonchus asper* Hoffm., prickly sow-thistle. *LEGUMINOSAE —Lathyrus odoratus* L., sweet pea; *Pisum sativum* L., pea.

Insusceptible species: *COMPOSITAE —Sonchus oleraceus* L., *S. arvensis* L., *Taraxacum officinale* Web., *Carduus arvensis* Curt. *CRUCIFERAE—Brassica oleracea* L. *CUCURBITACEAE—Cucumis sativus* L. *SOLANACEAE—Lycopersicon esculentum* Mill., *Nicotiana tabacum* L., *N. glutinosa* L., *Datura stramonium* L.

Geographical distribution: United States, England, Germany, Bermuda.

Induced disease: In lettuce varieties, clearing of veins followed by systemic chlorotic mottling, dwarfing and defective hearting; sometimes by scorching of leaf edges, vein necrosis or necrotic flecking between veins.

Transmission: By inoculation of expressed juice, in dilutions to 1:100 if mixed with a little 0.5 per cent sodium sulphite solution and a trace of powdered carborundum. By aphids, *Myzus persicae* (Sulz.) and *Macrosiphum gei* Koch (*APHIDIDAE*). Through seeds from diseased plants. It is believed that seed-borne virus is the most important source of primary inoculum in the spring.

Thermal inactivation: At 55 to 60° C in 10 minutes.

Filterability: Fails to pass L_1 Pasteur-Chamberland filter.

Literature: Ainsworth and Ogilvie, Ann. Appl. Biol., *26*, 1939, 279–297; Jagger, Jour. Agr. Res., *20*, 1921, 737–740; Newhall, Phytopath., *13*, 1923, 104–106.

17. **Marmor dahliae** H. (*loc. cit.*, 85). From New Latin *Dahlia*, generic name of host plant.

Common name: Dahlia-mosaic virus.

Hosts: *COMPOSITAE—Dahlia pinnata* Cav., dahlia. Experimentally, also *D. imperialis* Roezl.; *D. maxonii* Safford.

Geographical distribution: United States, Holland, Germany, England.

Induced disease: In intolerant varieties of dahlia, chlorotic mottling of foliage, leaf distortion, dwarfing of all stems and of roots, occasionally necrotic streaking of midveins. In tolerant varieties, inconspicuous chlorotic mottling or masked symptoms.

Transmission: By aphid, *Myzus persicae* (Sulz.) (*APHIDIDAE*). By grafting. Not by inoculation of expressed juice. Not through soil. Not through seeds from diseased plants.

Literature: Brierley, Am. Dahlia Soc. Bull., Ser. 9, No. 65, 1933; Contrib. Boyce Thompson Inst., *5*, 1933, 235–288; Goldstein, Bull. Torrey Bot. Club, *54*, 1927, 285–293.

18. **Marmor phaseoli** H. (*loc. cit.*, 87). From New Latin *Phaseolus*, generic name of bean.

Common name: Bean-mosaic virus.

Hosts: *LEGUMINOSAE—Phaseolus vulgaris* L., bean. Experimentally, also *Phascolus acutifolius* Gray var. *latifolius* Freem.; *P. aureus* Roxb.; *P. calcaratus* Roxb.; *P. lunatus* L.; *Lespedeza striata* (Thunb.) Hook. and Arn.; *Vicia faba* L.; *V. sativa* L., spring vetch.

Insusceptible species: *LEGUMINOSAE—Pisum sativum* L., garden pea; *Lathyrus odoratus* L., sweet pea.

Geographical distribution: World-wide, wherever beans are grown.

Induced disease: In bean, first leaves to be affected are crinkled, stiff, chlorotic; later leaves show chlorotic mottling; leaf margins often rolled down. Optimum temperature for expression of disease, 20 to 28° C, partial masking at 28 to 32° C, complete masking at 12 to 18° C.

Transmission: By inoculation of expressed juice in dilutions to 1:1000, using carborundum or other abrasive powder. By aphids, *Aphis rumicis* Linn., *Macrosiphum* (= *Illinoia*) *solanifolii* Ashm., *M. pisi* Kalt., *Aphis gossypii* Glov., *A. medicaginis* Koch, *A. spiraecola*, *Brevicoryne brassicae* (Linn.), *Hyalopterus atriplicis* Linn., *Macrosiphum ambrosiae* Thos., *Rhopalosiphum pseudobrassicae* Davis, and *Myzus persicae* (Sulz.) (*APHIDIDAE*). In beans, there is seed transmission to 30 to 50 per cent of plants grown from infected parents; pollen from infected plants is said to transmit virus.

Thermal inactivation: At 56 to 58° C in 10 minutes.

Literature: Fajardo, Phytopath., *20*, 1930, 469–494, 883–888; Murphy, *ibid.*, *30*, 1940, 779–784; Murphy and Pierce, *ibid.*, *28*, 1938, 270–273; Parker, Jour. Agr. Res., *52*, 1936, 895–915; Pierce, Phytopath., *24*, 1934, 87–115; Jour. Agr. Res., *49*, 1934, 183–188; *51*, 1935, 1017–1039; Reddick, II Congr. Intern. Path. Comp., 1931, 363–366; Reddick and Stewart, Phytopath., *8*, 1918, 530–534; Richards and Burkholder, Phytopath., *33*, 1943, 1215–1216; Wade and Andrus, Jour. Agr. Res., *63*, 1941, 389–393; Wade and Zaumeyer, U. S. Dept. Agr., Circ. 500, 1938; Walker and Jolivette, Phytopath., *33*, 1943, 778–788; Zaumeyer and Kearns, *ibid.*, *26*, 1936, 614–629; Zaumeyer and Wade, Jour. Agr. Res., *51*, 1935, 715–749.

19. **Marmor leguminosarum** H. (*loc. cit.*, 89). From New Latin *Leguminosae*, family name of pea.

Common name: Pea-mosaic virus.

Hosts: *LEGUMINOSAE—Lathyrus odoratus* L., sweet pea; *Pisum sativum* L., pea; *Trifolium pratense* L., red clover; *Vicia faba* L., broad bean. Experiment-

ally, also *Cicer arietinum* L.; *Desmodium canadense* (L.) DC.; *Lathyrus sativus* L., grass pea; *Lupinus albus* L., white lupine; *L. angustifolius*, blue lupine; *L. densiflorus* Benth.; *L. hartwegii* Lindl.; *L. nanus* Dougl.; *Medicago arabica* Huds., spotted bur clover; *M. hispida* Gaertn., toothed bur clover; *Melilotus alba* Desr., white sweet clover; *M. indica* All., annual yellow sweet clover; *M. officinalis* (L.) Lam., yellow sweet clover; *Phaseolus acutifolius* Gray, tepary bean; *P. vulgaris* L., bean; *Trifolium agrarium* L.; *T. carolinianum* Michx.; *T. dubium* Sibth.; *T. glomeratum* L., cluster clover; *T. hybridum* L., alsike clover; *T. incarnatum* L., crimson clover; *T. procumbens* L.; *T. reflexum* L.; *T. suaveolens*, Persian clover; *Vicia sativa* L., common vetch.

Insusceptible species: All tested species in families other than the *Leguminosae*.

Geographical distribution: United States, British Isles, Europe, New Zealand.

Induced disease: In pea, clearing of veins in young leaves, followed by chlorosis of newly formed leaves, stunting of plant, and systemic chlorotic mottling. In sweet pea, systemic chlorosis and chlorotic mottling, flower colors broken. In lupine, necrotic streak on one side of stem, stunting of plant and bending of growing point to injured side. Plant soon wilts and dies. In *Vicia faba*, mottled leaves contain characteristic isometric crystals in host-cell nuclei (especially within nucleoli) as well as in cell cytoplasm.

Transmission: By inoculation of expressed juice, with ease. By aphids, *Macrosiphum pisi* Kalt., *M. solanifolii* Ashm. (= *M. gei* Koch), and *Aphis rumicis* Linn. (*APHIDIDAE*). Not transmitted through seed.

Serological relationships: Specific precipitin reactions differentiate this virus from tobacco-mosaic virus, tobacco-etch virus, potato-mottle virus, potato mild-mosaic virus, potato aucuba-mosaic virus, and tobacco-ringspot virus.

Thermal inactivation: At 60° C in 10 minutes.

Literature: Chester, Phytopath., *25*, 1935, 686–701; Doolittle and Jones, *ibid.*, *15*, 1925, 763–772; Johnson and Jones, Jour. Agr. Res., *54*, 1937, 629–638; McWhorter, Phytopath., *31*, 1941, 760–761; Murphy and Pierce, *ibid.*, *27*, 1937, 710–721; Osborn, *ibid.*, *27*, 1937, 589–603; Pierce, Jour. Agr. Res., *51*, 1935, 1017–1039; Spierenburg, Tijdschr. Plantenz., *42*, 1936, 71–76; Zaumeyer and Wade, Jour. Agr. Res., *53*, 1936, 161–185.

20. **Marmor pisi** H. (*loc. cit.*, 90). From Latin *pisum*, pea.

Common name: Pea enation-mosaic virus.

Hosts: *LEGUMINOSAE—Pisum sativum* L., pea; *Vicia faba* L., broad bean. Experimentally, also *Lathyrus odoratus* L., sweet pea; *Soja max* (L.) Piper, soy bean; *Trifolium incarnatum* L., crimson clover.

Insusceptible species: *LEGUMINOSAE—Arachis hypogaea* L., peanut; *Medicago sativa* L., alfalfa; *Melilotus alba* Desr., white sweet clover; *M. officinalis* (L.) Lam., yellow sweet clover; *Phaseolus aureus* Roxb., mung bean; *P. vulgaris* L., bean; *Trifolium hybridum* L., alsike clover; *T. pratense* L., red clover; *T. repens* L., white Dutch clover. *SOLANACEAE—Lycopersicon esculentum* Mill., tomato; *Solanum tuberosum* L., potato.

Geographical distribution: United States, perhaps Germany.

Induced disease: In peas, systemic chlorotic mottling; in some varieties, as Alderman, occasional necrotic spots and numerous enations on lower surfaces of leaves. Pods distorted. In broad bean, systemic chlorotic spotting and striping of leaves. In sweet pea and soy bean, experimentally, systemic chlorotic mottling.

Transmission: By inoculation of expressed juice, using carborundum; more readily from aphid-inoculated plants than from mechanically-inoculated plants.

Infective in dilutions to 10^{-3}. By aphids, *Macrosiphum pisi* Kalt. and *M. solanifolii* Ashm. (= *M. gei* Koch) (*APHIDIDAE*), with incubation periods of about 12 hours before the insects can infect. Not through seeds from diseased plants.

Thermal inactivation: At 66° C in 10 minutes.

Literature: Böning, Forsch. Geb. Pflanzenkr. u. Immun. Pflanzenreich, *4*, 1927, 43–111; Johnson and Jones, Jour. Agr. Res., *54*, 1937, 629–638; Loring et al., Proc. Soc. Exp. Biol. and Med., *38*, 1938, 239–241; Osborn, Phytopath., *25*, 1935, 160–177; *28*, 1938, 749–754, 923–934; Pierce, Jour. Agr. Res., *51*, 1935, 1017–1039; Snyder, Phytopath., *24*, 1934, 78–80; Stubbs, *ibid.*, *27*, 1937, 242–266.

21. **Marmor medicaginis** II. (*loc. cit.*, 91). From New Latin *Medicago*, generic name of alfalfa (lucerne).

Common name: Alfalfa-mosaic virus.

Hosts: *LEGUMINOSAE—Medicago sativa* L., alfalfa (lucerne). *SOLANACEAE—Solanum tuberosum* L., potato. Experimentally, also transmissible to many species of dicotyledonous plants (summarized by Price, Am. Jour. Bot., *27*, 1940, 530–541) including *CUCURBITACEAE—Cucumis sativus* L., cucumber. *COMPOSITAE—Zinnia elegans* Jacq., zinnia. *LEGUMINOSAE—Phaseolus vulgaris* L., bean; *Trifolium incarnatum* L., crimson clover. *SOLANACEAE—Capsicum frutescens* L., pepper; *Lycopersicon esculentum* Mill., tomato; *Nicotiana tabacum* L., tobacco.

Geographical distribution: United States.

Induced disease: In alfalfa, systemic chlorotic mottling, tending to be masked at times. In bean, (most varieties) small necrotic primary lesions, reddish brown at periphery. No secondary lesions. Some bean varieties show no lesions after inoculation; one of these, Refugee Rogue, possesses two dominant genes either of which will confer this type of resistance. In tobacco, white necrotic flecks, small rings and arcs on inoculated leaves; later, systemic mottling, followed by production of necrotic oak-leaf patterns; virus content may be low in plants long diseased, especially in summer.

Transmission: By inoculation of expressed juice. By aphids, *Macrosiphum pisi* Kalt. (for typical strain) and *M. solanifolii* Ashm. (for potato-calico strain) (*APHIDIDAE*). Not through seeds from diseased plants.

Immunological relationships: Resistance to superinfection with the type of this virus is conferred by earlier infection with potato-calico virus (now considered a related strain but earlier regarded as distinct), but not by earlier infection with potato-mottle virus, cucumber-mosaic virus, or the Canada-streak strain of potato aucuba-mosaic virus.

Thermal inactivation: At 65 to 70° C in 10 minutes.

Other properties: Sedimentation constant, $73.9 \pm 5.2 \times 10^{-13}$ cm. per sec. in a unit centrifugal field. Specific volume 0.673. Particles spherical or nearly so. Diameter 16.5 millimicrons; weight 2.1×10^6 times hydrogen unit. Isoelectric point about pH 4.6. Inactivated and, more slowly, hydrolyzed by trypsin.

Literature: Black and Price, Phytopath., *30*, 1940, 444–447; Lauffer and Ross, Jour. Am. Chem. Soc., *62*, 1940, 3296–3300, Pierce, Phytopath., *24*, 1934, 87–115; Price, Am. Jour. Bot., *27*, 1940, 530–541; Ross, Phytopath., *31*, 1941, 394–410, 410–420; Wade and Zaumeyer, Jour. Am. Soc. Agron., *32*, 1940, 127–134; Zaumeyer, Jour. Agr. Res., *56*, 1938, 747–772.

Strains: At least one strain of alfalfa-mosaic virus was formerly considered as an independent virus, causing a disease known as calico in potato. It has now been given varietal rank and distinguished from the type, var. *typicum* Black and Price (Phytopath., *30*, 1940, 446) by the following name:

21a. *Marmor medicaginis* var. *solani* Black and Price (Phytopath., *30*, 1940,

446). From New Latin *Solanum*, generic name of potato.

Common name: Potato-calico strain of alfalfa-mosaic virus. Differing from the type by inducing a more severe disease in potato, in which it is commonly found in nature. (Price and Black, Phytopath., *30*, 1940, 444–447; Dykstra, *ibid.*, *29*, 1939, 917–933; Porter, Potato Assoc. Amer. Proc., *18*, 1931, 65–69; Hilgardia, *6*, 1931, 277–294; *9*, 1935, 383–394.)

22. **Marmor tulipae** H. (*loc. cit.*, 52). From New Latin *Tulipa*, generic name of tulip.

Common name: Tulip color-adding virus.

Hosts: *LILIACEAE—Tulipa gesneriana* L., garden tulip; *T. eichleri* Regel; *T. greigi* Regel.

Insusceptible species: *AMARYLLIDACEAE—Narcissus* sp., narcissus. *IRIDACEAE—Iris germanica* L., iris. *LILIACEAE—Allium cepa* L., onion. *SOLANACEAE—Nicotiana tabacum* L., tobacco.

Geographical distribution: Wherever hybrid tulips are grown.

Induced disease: In tulip, no obvious effect on leaves but dark striping of flower by pigment intensification. Little interference with growth of plant. No intracellular bodies.

Transmission: By hypodermic injections of expressed juice in dilutions to 10^{-5}. By aphids, *Myzus persicae* (Sulz.), *Macrosiphum solanifolii* Ashm. (= *M. gei* Koch, *Illinoia solanifolii* Ashm.), *Aphis* (= *Anuraphis*) *tulipae* B. de Fonsc. (on bulbs), and perhaps *Macrosiphum pelargonii* Kalt. (*APHIDIDAE*). Not through seeds from diseased plants.

Thermal inactivation: At 65 to 70° C in 10 minutes.

Literature: Hughes, Ann. Appl. Biol., *18*, 1931, 16–29; *21*, 1934, 112–119; McWhorter, Phytopath., *22*, 1932, 998 (Abst.); *25*, 1935, 898 (Abst.); Ann. Appl. Biol., *25*, 1938, 254–270.

23. **Marmor mite** H. (*loc. cit.*, 53). From Latin *mitis*, mild.

Common name: Lily latent-mosaic virus.

Hosts: *LILIACEAE—Lilium amabile; L. auratum* Lindl.; *L. canadense* L.; *L. candidum* L.; *L. cernuum; L. chalcedonicum* L.; *L. croceum* Chaix.; *L. davmottiae; L. elegans* Thunb.; *L. formosanum* Stapf.; *L. giganteum; L. henryi* Baker; *L. leucanthum; L. longiflorum* Thunb.; *L. myriophyllum; L. pumilum; L. regale* Wils.; *L. sargentiae* Wils.; *L. speciosum* Thunb.; *L. superbum* L.; *L. testaceum* Lindl.; *L. tigrinum* Ker; *L. umbellatum* Hort.; *L. wallacei; Tulipa gesneriana* L., garden tulip; *T. clusiana* Vent.; *T. linifolia* Regel.

Insusceptible species: *LILIACEAE—Allium cepa* L., onion; *Lilium hansoni* Leichtl. *IRIDACEAE—Iris germanica* L., iris. *SOLANACEAE—Nicotiana tabacum* L., tobacco.

Geographical distribution: Wherever lilies and tulips are cultivated.

Induced disease: In Easter lily, masked symptoms or systemic chlorotic mottling, in either case without necrotic flecking. In tulip, systemic chlorotic mottling in foliage and flower "breaking" (color removal, except in a few varieties in which color intensification occurs instead). Intracellular bodies characterize invaded tissues.

Transmission: By inoculation of expressed juice (rubbing surface of leaves), in both lily and tulip. By plugging and grafting of dormant bulbs of tulip. By aphids, *Myzus persicae* (Sulz.), *Macrosiphum solanifolii* Ashm. (= *M. gei* Koch), and *Aphis* (= *Anuraphis*) *tulipae* B. de Fonsc. (*APHIDIDAE*). Not through seeds from mosaic *Lilium longiflorum*.

Thermal inactivation: At 65 to 70° C in 10 minutes.

Literature: Atanasoff, Bull. Soc. Bot. Bulgarie, *2*, 1928, 51–60; Brierley, Phytopath., *29*, 1939, 3 (Abst.); *30*, 1940, 250–

257; *31*, 1941, 838–843; Brierley and Doolittle, *ibid.*, *30*, 1940, 171–174; Cayley, Ann. Appl. Biol., *15*, 1928, 529–539; *19*, 1932, 153–172; Guterman, Hort. Soc. N. Y., Yearbk., *1930*, 51–102; Hall, Gard. Chron., *93*, 1933, 330–331; Hughes, Ann. Appl. Biol,. *21*, 1934, 112–119; McKay and Warner, Nat. Hort. Mag., *12*, 1933, 179–216; McWhorter, Phytopath., *25*, 1935, 898 (Abst.); Science, *86*, 1937, 179; Ann. Appl. Biol., *25*, 1938, 254–270; Science, *88*, 1938, 411; Ogilvie and Guterman, Phytopath., *19*, 1929, 311–315.

24. **Marmor iridis** H. (*loc. cit.*, 55). From New Latin *Iris*, generic name of iris.

Common name: Iris-mosaic virus.

Hosts: *IRIDACEAE—Iris filifolia* Boiss., *I. tingitana* Boiss. and Reut., and *I. xiphium* L., bulbous irises; *Iris ricardi* Hort.; *I. unguicularis* Poir.; bearded iris, variety William Mohr.

Insusceptible species: *SOLANACEAE—Lycopersicon esculentum* Mill., tomato; *Nicotiana tabacum* L., tobacco; *Petunia hybrida* Vilm., petunia. *LILIACEAE—Tulipa gesneriana* L., tulip.

Geographical distribution: United States (Washington, Oregon, California), Holland, Bulgaria, France, England.

Induced disease: In bulbous irises, dwarfing of plant, chlorotic mottling of foliage, breaking of flowers. Rate of increase in planting stock decreased. Flower breaks usually darker than normal color of flower. Vacuolate intracellular bodies in some affected tissues.

Transmission: By injection of freshly extracted juice of diseased plants into internodal tissue. By aphids, *Macrosiphum* (= *Illinoia*) *solanifolii* Ashm. and *Myzus persicae* (Sulz.) (*APHIDIDAE*).

Literature: Brierley and McWhorter, Jour. Agr. Res., *53*, 1936, 621–635.

25. **Marmor sacchari** H. (*loc. cit.*, 60). From New Latin *Saccharum*, generic name of sugar cane, from Latin *saccharum*, sugar.

Common name: Sugar-cane mosaic virus.

Hosts: *GRAMINEAE—Saccharum officinarum* L., sugar cane; *Holcus sorghum* L., sorghum; *H. sudanensis* Bailey, Sudan grass; *Brachiaria platyphylla* Nash; *Chaetochloa magna* Scribn.; *C. verticillata* Scribn.; *Paspalum boscianum* Fluegge; *Syntherisma sanguinale* Dulac. Experimentally, also *Zea mays* L., corn (maize); *Chaetochloa lutescens* Stuntz; *Echinochloa crusgalli* Beauv.; *Miscanthus sinensis* Anderss., eulalia; *Panicum dichotomiflorum* Michx.; *Pennisetum glaucum* R. Br., pearl millet; *Saccharum narenga* Wall.

Insusceptible species: All tested species other than *Gramineae*.

Geographical distribution: Originally in Far East; now in nearly all countries where sugar cane is grown; believed still to be absent from Mauritius.

Induced disease: In sugar cane, systemic mottling chlorosis, light areas of pattern elongated, but crossing veins. Occasionally, stem cankers. Regularly, discoloration and necrosis in mature inner stalk tissues. Vacuolate intracellular bodies occur in diseased tissues. Canes sometimes recover, spontaneously losing the virus and becoming susceptible to reinfection.

Transmission: By inoculation of expressed juice (puncture through inoculum into young leaf). By aphids, *Aphis maidis* Fitch, *Carolinaia cyperi* Ainslie, *Hysteroneura setariae* (Thomas), and *Toxoptera graminum* Rond.; not by *Sipha flava* Forbes (*APHIDIDAE*). Not by *Draeculacephala mollipes* (Say) (*CICADELLIDAE*).

Serological relationships: Specific neutralizing and precipitating antibodies have been demonstrated.

Thermal inactivation: At 53 to 54° C in 10 minutes in leaf tissues.

Other properties: Active after storage 27 days at −6° C.

Literature: Brandes, Jour. Agr. Res., *19*, 1920, 131–138, 517–522; *24*, 1923, 247–262; Desai. Current Science, *3*, 1935, 18; Forbes and Mills, Phytopath., *33*, 1943, 713–718; Ingram and Summers, Jour. Agr. Res., *52*, 1936, 879–888; Kunkel, Bull. Exp. Sta. Hawaiian Sugar Planters' Assoc., Bot. Ser., *3*, 1924, 115–167; Matz, Jour. Agr. Res., *46*, 1933, 821–839; Rafay, Indian Jour. Agr. Science *5*, 1935, 663–670; Seín, Jour. Dept. Agr. Porto Rico, *14*, 1930, 49–68; Stoneberg, U. S. Dept. Agr., Tech. Bull. 10, 1927; Tate and Vandenberg, Jour. Agr. Res., *59*, 1939, 73–79.

26. **Marmor cepae** H. (*loc. cit.*, 66). From Latin *cepa*, onion.

Common name: Onion yellow-dwarf virus.

Host: *LILIACEAE—Allium cepa* L., onion (the variety *viviparum* Metz. is symptomless when infected and may serve as an unrecognized reservoir of virus).

Geographical distribution: United States, Germany, Czecho-Slovakia, Russia, New Zealand.

Induced disease: In onion (most varieties), yellow streaks at base of developing leaf, followed by yellowing, crinkling, and flattening of newly formed leaves; leaves prostrate, flower stalks bent, twisted, stunted; plants reduced in size, bulbs small, yield of seeds reduced. A few varieties of onion are relatively tolerant, and the tree-onion, var. *viviparum* is symptomless after infection.

Transmission: By inoculation of expressed juice. By 48 of 51 tested species of aphid, principally *Aphis rumicis* Linn., *A. maidis* Fitch, and *Rhopalosiphum prunifoliae* Fitch (*APHIDIDAE*). Not through seeds from diseased plants. Not by contaminated soil.

Thermal inactivation: At 75 to 80° C in 10 minutes.

Other properties: Virus withstands dilution to 10^{-4}, storage at 29° C for about 100 hours and storage at −14° C for more than time tested (6 hours), but is inactivated by drying in leaf tissues.

Literature: Andreyeff, Rev. Appl. Mycol., *17*, 1938, 575–576; Blattny, Ochrana Rostlin, *10*, 1930, 130–138; Bremer, Phytopath. Ztschr., *10*, 1937, 79–105; Brierley and Smith, Phytopath., *34*, 1944, 506–507; Chamberlain and Baylis, New Zealand Jour. Science and Technology, *21*, 1939, 229A–236A; Drake et al., Iowa State Coll. Jour. Science, *6*, 1932, 347–355; Jour. Econ. Ent., *26*, 1933, 841–846; Henderson, Phytopath., *20*, 1930, 115 (Abst.); Iowa State Coll., Research Bull. *188*, 1935, 211–255; Melhus et al., Phytopath., *19*, 1929, 73–77; Porter, U. S. Dept. Agr., Plant Dis. Rept., *12*, 1928, 93; Tate, Iowa State Coll. Jour. Science, *14*, 1940, 267–294.

27. **Marmor scillearum** Smith and Brierley (Phytopath., *34*, 1944, 503.) From New Latin *Scilleae*, name of tribe in which hosts are classed.

Common name: Ornithogalum-mosaic virus.

Hosts: *LILIACEAE* (of the tribe Scilleae)—*Ornithogalum thyrsoides* Jacq.; probably also *Galtonia candicans* Decne.; *Hyacinthus orientalis* L., hyacinth; *Lachenalia* sp.

Insusceptible species: *LILIACEAE* (of the tribe Scilleae)—*Muscari botryoides* Mill.; *Scilla peruviana* L.; *Camassia leichtlinii* (Baker) S. Wats.; *Hyacinthus azureus* (Fenzl.) Baker. *AMARYLLIDACEAE—Pancratium maritimum; Zephyranthus* sp. *IRIDACEAE—Tritonia crocata* (L.) Ker. *LILIACEAE—Agapanthus africanus; Allium cepa*, onion; *A. cernuum* Roth.; *A. fistulosum* L.; *A. porrum* L.; *Gloriosa rothschildiana* O'Brien; *Lilium formosanum* Stapf.; and *L. longiflorum*. *SOLANACEAE—Nicotiana tabacum* L.

Geographical distribution: United States (Oregon; probably also Alabama and presumed to be widespread in plants of the squill tribe, *Scilleae*, of the family *LILIACEAE*).

Induced disease: In *Ornithogalum thyrsoides*, young leaves finely mottled with light and dark green, and becoming more conspicuously mottled with gray or yellow as the leaves mature; flower stalks sometimes boldly marked with light and dark green blotches. In perianth segments, thin longitudinal streaks.

Transmission: By inoculation of expressed juice in the presence of fine carborundum powder, with difficulty. By aphids, *Aphis gossypii* Glov., *Macrosiphum lilii* Monell, *M. solanifolii* Ashm., and *Myzus persicae* (Sulz.); less efficiently by *Myzus circumflexus* (Buckt.) (*APHIDIDAE*).

Key to the species of the Miscellaneous Mosaic-Virus Group.

Many of the following viruses, although described in some detail in the literature, stand in need of reinvestigation to determine additional properties and possible relationships to preceding groups.

I. Affecting species of *MALVACEAE*.
28. *Marmor abutilon.*

II. Affecting species of *CELASTRACEAE*.
29. *Marmor euonymi.*

III. Affecting species of *OLEACEAE*.
30. *Marmor ligustri.*

IV. Affecting species of *LEGUMINOSAE* (and no. 39, other families also).
31. *Marmor laburni.*
32. *Marmor arachidis.*
33. *Marmor trifolii.*
34. *Marmor pachyrhizi.*
35. *Marmor vignae.*
36. *Marmor repens.*
37. *Marmor fastidiens.*
38. *Marmor iners.*
39. *Marmor efficiens.*

V. Affecting species of *GRAMINEAE*.
40. *Marmor tritici.*
41. *Marmor graminis.*

VI. Affecting species of *MUSACEAE*.
42. *Marmor abaca.*

VII. Affecting species of *PASSIFLORACEAE*.
43. *Marmor passiflorae.*

VIII. Affecting species of *ROSACEAE*.
44. *Marmor flaccumfaciens.*
45. *Marmor rosae.*
46. *Marmor veneniferum.*
47. *Marmor mali.*
48. *Marmor fragariae.*
49. *Marmor marginans.*
50. *Marmor rubi.*
51. *Marmor persicae.*
52. *Marmor astri.*
53. *Marmor rubiginosum.*
54. *Marmor cerasi.*
55. *Marmor lineopictum.*
56. *Marmor pallidolimbatus.*
57. *Marmor nerviclarens.*

IX. Affecting species of *VITACEAE*.
58. *Marmor viticola.*
X. Affecting species of *SANTALACEAE*.
59. *Marmor santali.*
XI. Affecting species of *CONVOLVULACEAE* and, experimentally, of other families.
60. *Marmor secretum.*
XII. Affecting species of *GERANIACEAE*.
61. *Marmor pelargonii.*
XIII. Affecting species of *SOLANACEAE* and in most cases also of other families.
62. *Marmor angliae.*
63. *Marmor aevi.*
64. *Marmor raphani.*
XIV. Affecting species of *PRIMULACEAE*.
65. *Marmor primulae.*
XV. Affecting species of *MORACEAE*.
66. *Marmor caricae.*
XVI. Affecting species of *RUTACEAE*.
67. *Marmor italicum.*

28. **Marmor abutilon** H. (*loc. cit.*, 50). From New Latin *Abutilon*, generic name of a host.

Common name: Abutilon-mosaic virus.

Hosts: *MALVACEAE—Abutilon striatum* Dicks. var. *thompsonii* Veitch. Experimentally, also *Abutilon arboreum* Sweet; *A. avicennae* Gaertn.; *A. esculentum* St. Hil.; *A. indicum* Sweet; *A. insigne* Planch.; *A. megapotamicum* St. Hil. and Naud.; *A. regnellii* Miq.; *A. sellowianum* Regel; *A. venosum* Lem.; *A. vitifolium* Presl.; *Althaea ficifolia* Cav.; *A. officinalis* L.; *A. rosea* Cav.; *Anoda hastata* Cav.; *Kitaibelia vitifolia* Willd.; *Malva borealis; M. crispa; M. mauritiana* Mill.; *M. sylvestris* L.; *M. verticillata* L.; *Malvastrum capense* Garcke; *Modiola decumbens* G. Don.; *Sida mollis* Herb.; *S. napaea* Cav.; *Sidalcea candida* A. Gray.

Insusceptible species: *MALVACEAE—Althaea taurinensis; Sidalcea purpurea; Sphaeralcea umbellata* G. Don.

Geographical distribution: Germany, France, England, United States; originally obtained from a single variegated seedling found among green plants of *Abutilon striatum* imported from the West Indies in 1868 by Veitch and Sons; subsequently the infected plant was propagated vegetatively as an ornamental variety.

Induced disease: In *Abutilon*, systemic chlorotic mottling. Recovery occurs if there is persistent removal of affected leaves, suggesting that the virus does not increase in stems. After recovery, plants are susceptible to reinfection.

Transmission: By grafting, except patch-bark-grafting, which is ineffective. Occasionally through seeds from diseased plants. Not by inoculation of expressed juice. No insect vector is known.

Varieties: Distinctive strains have been noted, but not separately named; one isolate originally occurring in *Abutilon darwini* var. *tesselatum*, seems to belong here; it differs from the type principally in severity of induced disease and in ability to infect *Lavatera arborea*.

Literature: Baur, Ber. d. Deutsch. Bot. Gesellsch., *22*, 1904, 453–460; *24*, 1906, 416–428; *25*, 1907, 410–413; K. Preuss. Akad. Wiss., Sitzungsber., 1906, 11–19; Davis, Ann. Missouri Bot. Gard., *16*, 1929, 145–226; Hertzsch, Ztschr. f. Bot., *20*, 1927, 65–85; Keur, Phytopath., *23*, 1933, 20 (Abst.); *24*, 1934, 12–13 (Abst.); Bull. Torrey Bot. Club, *61*, 1934, 53–70; Lindemuth, Gartenflora, *51*, 1902, 323–326.

29. **Marmor euonymi** H. (*loc. cit.*, 51). From New Latin *Euonymus*, generic name of host.

Common name: Euonymus-mosaic virus.

Hosts: *CELASTRACEAE—Euonymus japonica* L. f. (sometimes written *Evonymus japonicus*). Probably also *E. radicans* Sieb.

Geographical distribution: Germany.

Induced disease: In *Euonymus japonica*, persistent yellowing along veins.

Transmission: By grafting.

Literature: Baur, Ber. d. Deutsch. Bot. Gesellsch., *26a*, 1908, 711–713; Rischkow, Biol. Zentralbl., *47*, 1927, 752–764.

30. **Marmor ligustri** H. (*loc. cit.*, 52). From New Latin *Ligustrum*, generic name of host, from Latin *ligustrum*, ancient name of privet plant.

Common name: Ligustrum-mosaic virus.

Host: *OLEACEAE—Ligustrum vulgare* L., common privet.

Geographical distribution: Germany.

Induced disease: Systemic chlorotic spotting.

Transmission: By grafting. Not through seeds from diseased plants.

Literature: Baur, Ber. d. Deutsch. Bot. Gesellsch., *25*, 1907, 410–413.

31. **Marmor laburni** H. (*loc. cit.*, 51). From generic name of a host plant, *Laburnum vulgare*.

Common name: Laburnum-mosaic virus.

Hosts: *LEGUMINOSAE—Laburnum vulgare Griseb.* (= *L. anagyroides* Medic.), bean tree. Experimentally, also *Cytisus hirsutus* L.

Insusceptible species: *LEGUMINOSAE—Laburnum alpinum* Griseb.; *Cytisus purpureus*.

Geographical distribution: Germany.

Induced disease: Systemic chlorotic variegation.

Transmission: By bark grafts or by budding. Not through seeds from diseased plants of *Laburnum vulgare*.

Literature: Baur, Ber. d. Deutsch. Bot. Gesellsch., *25*, 1907, 410–413.

32. **Marmor arachidis** H. (*loc. cit.*, 67). From New Latin *Arachis*, generic name of peanut.

Common name: Peanut-rosette virus.

Host: *LEGUMINOSAE—Arachis hypogaea* L., peanut.

Geographical distribution: Union of South Africa, Madagascar, Tanganyika Territory, Uganda, Senegal, Gambia, Sierra Leone, Java.

Induced disease: In peanut, yellowing of young leaves, at first with green veins; reduction in leaf size, petiole length, and internode length, producing rosette; curling and distortion of later-formed, wholly chlorotic or chlorotically mottled leaflets. Seed formation inhibited. No abnormal proliferation of tissues.

Transmission: By grafting. By both winged and wingless individuals of the aphid, *Aphis laburni* Kalt. (= *A. leguminosae* Theob.) (*APHIDIDAE*). Not by 13 tested species of leafhoppers. Not by inoculation of expressed juice. Not through seed from diseased plants. Not through soil.

Literature: Hayes, Trop. Agr., *9*, 1932, 211–217; McClintock, Science, *45*, 1917, 47–48; Soyer, Publ. Inst. Nat. Étud. Agron. Congo Belge, Sér. Sci., *21*, 1939, 23 pages (Rev. Appl. Mycol., *19*, 1940, 386, Abst.); Storey and Bottomley, Ann. Appl. Biol., *15*, 1928, 26–45; Zimmerman, Der Pflanzer, *3*, 1907, 129–133; *9*, 1913, 59–63.

33. **Marmor trifolii** H. (*loc. cit.*, 93). From New Latin *Trifolium*, generic name of red clover, from Latin *trifolium*, clover.

Common name: Red-clover vein-mosaic virus.

Hosts: *LEGUMINOSAE—Trifolium pratense* L., red clover; *Lathyrus odoratus* L., sweet pea; *Vicia faba* L., broad bean. Experimentally, also *Trifolium hybridum* L., alsike clover; *T. incarnatum* L., crim-

son clover; *T. repens* L., white clover; *Melilotus alba* Desr., white sweet clover; *Pisum sativum* L., pea.

Insusceptible species: *LEGUMINOSAE—Phaseolus vulgaris* L., bean; *P. aureus* Roxb., mung bean; *Medicago sativa* L., alfalfa. *SOLANACEAE—Lycopersicon esculentum* Mill., tomato; *Nicotiana tabacum* L., tobacco; *N. glutinosa* L.; *N. langsdorffii* Weinm.; *N. rustica* L.; *N. sylvestris* Spegaz. and Comes; *Solanum tuberosum* L., potato.

Geographical distribution: United States.

Induced disease: In red clover, yellow color along veins, but no mottling. Sometimes small yellow spots in interveinal areas. Little or no stunting. In *Vicia faba*, experimentally, necrotic splotches or rings sometimes at site of inoculation. Clearing of veins followed by appearance of whitish bands along the veins. Stalks discolored, purplish. Diseased plants are stunted and often die back to a point near the base of the stalk, inducing new growth from buds on the stem.

Transmission: By inoculation of expressed juice, using carborundum. By aphid, *Macrosiphum pisi* Kalt. (*APHIDIDAE*), without incubation period and without long retention. Not by aphids, *Macrosiphum solanifolii* Ashm. (= *M. gei* Koch) or *Aphis rumicis* Linn. (*APHIDIDAE*).

Thermal inactivation: At 60° C in 10 minutes.

Literature: Osborn, Phytopath., *27*, 1937, 1051–1058; Zaumeyer, Jour. Agr. Res., *56*, 1938; 747–772; Zaumeyer and Wade, Phytopath., *27*, 1937, 1009–1013.

34. **Marmor pachyrhizi** *spec. nov.* From New Latin *Pachyrhizus*, generic name of sincamas.

Common name: Sincamas-mosaic virus.

Host: *LEGUMINOSAE—Pachyrhizus erosus* (L.) Urb., sincamas (yam bean).

Insusceptible species: *LEGUMINOSAE—Phaseolus vulgaris* L., bean.

Geographical distribution: Philippine Islands.

Induced disease: In sincamas, chlorotic mottling of foliage; in plants infected when young, dwarfing.

Transmission: By inoculation of expressed juice, in the presence of sand as abrasive. Through about 25 percent of the seeds from infected plants. Not through soil, interlacing of roots, or casual contacts of leaves and stems. No insect vector is known.

Literature: Fajardo and Marañon, Philippine Jour. Science, *48*, 1932, 129–142.

35. **Marmor vignae** *spec. nov.* From New Latin *Vigna*, generic name of cowpea, from family name of an Italian botanist, Domenico Vigna.

Common name: Cowpea-mosaic virus.

Hosts: *LEGUMINOSAE—Vigna sinensis* (L.) Endl., cowpea. Experimentally, also *Phaseolus lunatus* L., lima bean.

Geographical distribution: United States (Arkansas, Oklahoma, Louisiana, Indiana, Georgia, Iowa, Mississippi, Kansas, New Jersey).

Induced disease: In cowpea, clearing of veins followed by chlorotic mottling, slight convex cupping of leaflets, shortened internodes, abortion of flowers, twisting of petioles, delayed maturity. Malformation of leaves, stunting of plants, and reduction of yield more pronounced in some varieties of cowpea than in others.

Transmission: By inoculation of expressed juice, especially in the presence of fine carborundum powder. By aphids, *Macrosiphum solanifolii* Ashm., *M. pisi* Kalt., *Aphis gossypii* Glov. (*APHIDIDAE*); not by various beetles nor by the bean leafhopper, *Empoasca fabae* LeB. (*CICADELLIDAE*). Through 5 per cent of seeds from infected cowpea plants.

Thermal inactivation: At 72 to 75° C in 10 minutes.

Other properties: Infectious in dilutions as high as 1:1000 and after 2 days

storage in expressed juice at room temperature, 20 to 25° C.

Literature: Elliott, Phytopath., *11*, 1921, 146–148; Gardner, Indiana Acad. Science Proc., *36*, 1927, 231–247; *37*, 1928, 417; McLean, Phytopath., *31*, 1941, 420–430; Smith, Science, *60*, 1924, 268.

36. **Marmor repens** Johnson. (Phytopath., *32*, 1942, 114.) From Latin *repens*, unlooked for, in reference to unexpected discovery of this virus as a constituent of a complex formerly regarded as a single virus, so-called "white-clover mosaic virus".

Common name: Pea-wilt virus.

Hosts: *LEGUMINOSAE—Trifolium repens* L., white clover. Experimentally, also *Lathyrus odoratus* L., *Lens esculenta* Moench.; *Lupinus albus* L.; *Medicago lupulina* L.; *Melilotus alba* Desr.; *Phaseolus aureus* Roxb., mung bean; *P. vulgaris* L., bean; *Pisum sativum* L., pea; *Trifolium hybridum* L.; *T. incarnatum* L.; *T. pratense* L.; *Vicia faba* L.; *V. sativa* L.; *Vigna sinensis* (L.) Endl., cowpea.

Insusceptible species: *CARYOPHYLLACEAE—Stellaria media* (L.) Cyrill. *CHENOPODIACEAE—Beta vulgaris* L.; *Spinacia oleracea* L. *COMPOSITAE—Callistephus chinensis* Nees; *Lactuca sativa* L.; *Taraxacum officinale* Weber; *Zinnia elegans* Jacq. *CRUCIFERAE—Barbarea vulgaris* R. Br.; *Brassica oleracea* L.; *Raphanus sativus* L. *CUCURBITACEAE—Cucumis sativus* L. *GRAMINEAE—Zea mays* L. *LEGUMINOSAE—Glycine max* Merr.; *Lupinus hirsutus* L.; *Medicago sativa* L. *LILIACEAE—Lilium formosanum* Stapf. *PLANTAGINACEAE—Plantago lanceolata* L.; *P. major* L. *POLYGONACEAE—Rumex acetosella* L. *SCROPHULARIACEAE — Antirrhinum majus* L. *SOLANACEAE—Datura stramonium* L.; *Lycopersicon esculentum* Mill.; *Nicotiana glutinosa* L.; *N. rustica* L.; *N. sylvestris* Spegaz. and Comes; *N. tabacum* L.; *Solanum nigrum* L.

Geographical distribution: United States (Washington).

Induced disease: In white clover, systemic chlorotic mottling. In pea, experimentally, originally infected leaves wilt and die, remaining attached to the stem by their shriveled petioles; a few adjacent lower leaves may also wilt and die; in most varieties the top foliage remains green, but in two varieties, Alaska and Canada White, it mottles faintly; stems show faint grayish discoloration; plants are retarded in growth and dwarfed. If pea-mottle virus, *Marmor efficiens* Johnson, is also present, a severe streak disease occurs. Intracellular inclusions absent. In mung bean, experimentally, necrotic zonate local lesions. In cowpea, experimentally, brown necrotic local lesions in inoculated primary leaves, diffuse areas of bleaching in uninoculated trifoliate leaves. In bean, experimentally, mild chlorotic mottling except in three varieties that appear insusceptible (varieties Ideal Market, Kentucky Wonder, and Navy Robust).

Transmission: By inoculation of expressed juice. Not by dodder, *Cuscuta campestris* Yunck. (*CONVOLVULACEAE*). Not by pea aphid, *Macrosiphum pisi* Kalt. (*APHIDIDAE*). No insect vector is known.

Thermal inactivation: At 58 to 60° C in 10 minutes.

Filterability: Passes Berkefeld W filter candle.

Other properties: Infectious in dilution of 1:100,000. Not inactivated by storage in juice of infected plants at about 25° C for one month or by similar storage in dried tissues of infected pea plants.

Literature: Johnson, Phytopath., *32*, 1942, 103–116; Pierce, Jour. Agr. Res., *51*, 1935, 1017–1039.

37. **Marmor fastidiens** *spec. nov.* From Latin *fastidiens*, disdaining, in reference to slight irregularities in the reported host ranges of constituent strains and failure of this virus to infect certain varieties of the pea although it

may utilize many other varieties of this species as host.

Common name: Alsike-clover mosaic virus.

Hosts: *LEGUMINOSAE—Trifolium hybridum* L., alsike clover; *Pisum sativum* L., pea (except the varieties Horal, Perfection, and Surprise). Experimentally, also *Crotalaria striata* DC.; *C. retusa* L.; and *C. spectabilis* Roth (the two last-named species are reported to be insusceptible to the type strain of the virus, but susceptible to one or more of the other tested strains); *Lupinus albus* L.; *L. angustifolius* L.; *Medicago sativa* L.; *Melilotus alba* Desr.; *Phascolus vulgaris* L., bean; *Trifolium incarnatum* L.; *T. pratense* L.; *Vicia faba* L.

Insusceptible species: *SOLANACEAE—Datura stramonium* L.; *Nicotiana glauca* Graham; *N. glutinosa* L.; *N. tabacum* L.; *Petunia hybrida* Vilm. *LEGUMINOSAE—Phaseolus aureus* Roxb., mung bean; *P. lunatus* L., sieva bean; *Soja max* (L.) Piper, soybean; *Trifolium repens* L., white clover; *Vicia sativa* L., spring vetch.

Induced disease: In pea and bean, experimentally, systemic chlorotic mottling; some isolates kill inoculated leaves and even cause death of infected plants.

Transmission: By inoculation with expressed juice, at dilutions to 1:6000 or 1:8000. No insect vector is known.

Thermal inactivation: At 60 to 65° C in 10 minutes; one strain at lower temperature, 54 to 58° C.

Strains: Several strains have been distinguished by the severity of their effects on host plants. These may be characterized as follows: var. *fastidiens*, *var. nov.*, type variety, the first of the strains to be described (originally known as alsike clover mosaic virus 1), induces mild disease in pea, does not infect red clover; var. *mite*, *var. nov.*, described as pea mosaic virus 4, induces mild symptoms on pea, infects red clover; var. *reprimens*, *var. nov.*, described as pea mosaic virus 5, stunts peas severely; var. *denudans*, *var. nov.*, described as alsike clover mosaic virus 2, defoliates pea plants. Varietal names from New Latin *fastidiens*, epithet of the species, and from Latin *mitis*, mild; *reprimere*, to restrain; and *denudare*, to denude; all three in reference to induced symptoms.

Literature: Wade and Zaumeyer, Phytopath., *28*, 1938, 505–511; Zaumeyer, Jour. Agr. Res., *60*, 1940, 433–452.

38. **Marmor iners** *spec. nov.* From Latin *iners*, sluggish or inert, in reference to failure of the virus to spread systemically in certain of its hosts.

Common name: Pea-streak virus.

Hosts: *LEGUMINOSAE—Pisum sativum* L., pea. Experimentally, also *Galega officinalis* L., goat's rue; *Glycine soja* Sieb. and Zucc., soya bean; *Lathyrus odoratus* L., sweet pea; *Lotus hispidus* Desf.; *Lupinus angustifolius* L., blue lupin; *L. luteus* L., yellow lupin; *L. mutabilis* Sweet; *Phaseolus vulgaris* L., bean; *Trifolium arvense* L., haresfoot trefoil; *T. cernuum* Brot., nodding clover; *T. fragiferum* L., strawberry clover; *T. glomeratum* L., cluster clover; *T. hybridum* L., alsike clover; *T. pratense* L., red clover; *T. repens* L., white clover; *Vicia villosa* Roth., hairy vetch. *CUCURBITACEAE—Cucumis melo* L., rock melon; *C. sativus* L., cucumber; *Cucurbita pepo* L., marrow.

Insusceptible species: *CHENOPODIACEAE—Spinacia oleracea* L., spinach; *Beta vulgaris* L., beet. *COMPOSITAE—Calendula officinalis* L., calendula; *Lactuca sativa* L., lettuce; *Zinnia elegans* Jacq., zinnia. *CRUCIFERAE—Brassica napus* L., swede; *B. oleracea* L., cabbage; *B. rapa* L., turnip; *Matthiola incana* R. Br., stock; *Raphanus sativus* L., radish; *Sisymbrium officinale* (L.) Scop., hedge mustard. *LEGUMINOSAE—Arachis hypogaea* L., peanut; *Lathyrus latifolius* L., perennial sweet pea; *L. pubescens* Hook. and Arn., Argentine sweet pea; *Lotus corniculatus* L.; *Lupinus arboreus* Sims, tree lupin; *Medicago arabica* Huds.; *M. sativa* L., lucerne (alfalfa); *Phaseolus multiflorus* Willd., run-

ner bean; *Trifolium striatum* L., striated clover; *T. subterraneum* L., subterranean clover; *Vicia faba* L., broad bean. *PLANTAGINACEAE — Plantago lanceolata* L., plantain. *SCROPHULARIACEAE—Antirrhinum majus* L. *SOLANACEAE—Cyphomandra betacea* Sendt., tree tomato; *Datura stramonium* L., Jimson weed; *Nicotiana glauca* R. Grah.; *N. rustica* L., Turkestan tobacco; *N. tabacum* L., tobacco; *Physalis peruviana* L., Cape gooseberry; *Solanum nigrum* L., black nightshade. *TROPAEOLACEAE—Tropaeolum majus* L., nasturtium. *UMBELLIFERAE—Apium graveolens* L., celery.

Geographical distribution: New Zealand.

Induced disease: In the pea, stunting, wilting of young leaves, purple or purple-brown spotting on young leaves, dark streak on stem. Near tip, stem may die. Stem becomes brittle, tip bent to one side. Pods may remain flat and turn dark purple or purple-brown, or if already formed may show purple or purple-brown markings. Older leaves turn yellow, then brown and shrivelled. Infected plants usually die within two or three weeks. In inoculated plants small brown primary lesions, rapidly increasing in size especially along veins, eventually involve the whole leaf; petiole and stem streak follows. Among garden peas, the varieties Pride of the Market, Little Marvel, Wm. Massey and Autocrat are little affected; among field peas, the varieties Unica and White Ivory are equally resistant. In cucumber, experimentally, numerous brown, necrotic local lesions, each with light colored center and surrounding light-yellow halo. In bean, experimentally, local and systemic necrosis, stem streak, death of plant.

Transmission: By inoculation of expressed juice, best with an abrasive powder such as fine sand. Not by *Myzus persicae* (Sulz.), *Macrosiphum solani* (*APHIDIDAE*), nor *Thrips tabaci* Lind. (*THRIPIDAE*). No insect vector is known.

Thermal inactivation: At 78 to 80° C in 10 minutes.

Filterability: Passes Mandler filters of preliminary, regular, and fine grades.

Other properties: Dilution end point $1:10^6$. Not inactivated at room temperature in 41 days.

Literature: Chamberlain, New Zealand Jour. Science and Technology, *20*, 1939, 365A-381A.

39. **Marmor efficiens** Johnson. (Phytopath., *32*, 1942, 114.) From Latin *efficiens*, effective, in reference to ability of this virus to cause mottling in all tested varieties of pea in contrast with inability of pea-wilt virus, a second constituent of the complex earlier known as "white-clover mosaic virus," to produce such chlorotic symptoms in tested varieties other than Alaska and Canada White.

Common name: Pea-mottle virus.

Hosts: *LEGUMINOSAE—Trifolium repens* L., white clover; *Pisum sativum* L., pea. Experimentally, also *CARYOPHYLLACEAE—Stellaria media* (L.) Cyrill. *CHENOPODIACEAE—Spinacia oleracea* L., spinach. *CUCURBITACEAE—Cucumis sativus* L. *LEGUMINOSAE—Lathyrus odoratus* L.; *Lens esculenta* Moench.; *Lupinus albus* L.; *L. hirsutus* L.; *Medicago lupulina* L.; *M. sativa* L., alfalfa (lucerne); *Melilotus alba* Desr.; *Phaseolus aureus* Roxb.; *P. vulgaris* L., bean; *Trifolium hybridum* L.; *T. incarnatum* L.; *T. pratense* L.; *Vicia faba* L.; *V. sativa* L. *SCROPHULARIACEAE—Antirrhinum majus* L.

Insusceptible species: *CHENOPODIACEAE—Beta vulgaris* L., sugar beet. *COMPOSITAE—Callistephus chinensis* Nees; *Lactuca sativa* L.; *Taraxacum officinale* Weber; *Zinnia elegans* Jacq. *CRUCIFERAE—Barbarea vulgaris* R. Br.; *Brassica oleracea* L.; *Raphanus sativus* L. *GRAMINEAE—Zea mays* L. *LEGUMINOSAE—Glycine max* Merr.; *Vigna sinensis* (L.) Endl. *LILIACEAE—Lilium formosanum* Stapf. *PLANTAGINACEAE — Plantago*

lanceolata L.; *P. major* L. *POLYGONACEAE—Rumex acetosella* L. *SOLANACEAE—Datura stramonium* L.; *Lycopersicon esculentum* Mill.; *Nicotiana glutinosa* L.; *N. rustica* L.; *N. sylvestris* Spegaz. and Comes; *N. tabacum* L.; *Solanum nigrum* L.

Geographical distribution: United States (Washington).

Induced disease: Experimentally, in pea, developing leaves late in opening; clearing of veins, chlorotic spotting, stunting, chlorotic mottling; stipules mottled; stems, pods, and seeds appear normal. If pea-wilt virus (*Marmor repens* Johnson) is also present, a severe streak disease occurs. Intracellular inclusions absent. In bean, light yellow spots and clearing of veins. In spinach, severe chlorotic mottling, dwarfing. In alfalfa, streaks of yellowing along veins, chlorotic mottling.

Transmission: By inoculation of expressed juice. By dodder, *Cuscuta campestris* Yunck. (*CONVOLVULACEAE*). Not by pea aphid, *Macrosiphum pisi* Kalt. (*APHIDIDAE*). No insect vector is known.

Thermal inactivation: At 60 to 62° C in 10 minutes.

Filterability: Passes Berkefeld W filter candle.

Other properties: Infectious in dilution of 1:10,000 and after storage in expressed juice or dried tissues for one month at about 25° C.

Literature: Johnson, Phytopath., *32*, 1942, 103–116; Johnson and Jones, Jour. Agr. Res., *54*, 1937, 629–638; Pierce, *ibid.*, *51*, 1935, 1017–1039; Zaumeyer and Wade, *ibid.*, *51*, 1935, 715–749.

40. **Marmor tritici** H. (*loc. cit.*, 61). From Latin *triticum*, wheat.

Common names: Wheat-mosaic virus, wheat-rosette virus.

Hosts: *GRAMINEAE—Triticum aestivum* L., wheat; *Secale cereale* L., rye. Experimentally, also all tested species of the tribe Hordeae; *Triticum compactum* Host; *T. turgidum* L.; *T. durum* Desf.; *T. dicoccum* Schrank; *T. spelta* L.; *T. polonicum* L.; *T. monococcum* L., *Hordeum vulgare* L., barley.

Insusceptible species: *GRAMINEAE—Bromus inermis* Leyss., awnless bromegrass (of the tribe *Festuceae*).

Geographical distribution: United States, Japan.

Induced disease: In wheat, systemic chlorotic mottling, with dwarfing in some varieties; vacuolate, rounded intracellular bodies in diseased cells, usually close to nucleus. Some selections of Harvest Queen wheat are resistant.

Transmission: Through soil; remains infectious in soil 6 or more years. By inoculation of expressed juice (needle punctures in stem). Not through seeds or stubble of diseased plants. No insect vector is known.

Thermal inactivation: Contaminated soil becomes incapable of infecting wheat plants if heated for 10 minutes at 60° C though not if heated for the same length of time at 50° C.

Literature: Johnson, Science, *95*, 1942, 610; McKinney, Jour. Agr. Res., *23*, 1923, 771–800; U. S. Dept. Agr., Bull. 1361, 1925; U. S. Dept. Agr., Circ. 442, 1937; Jour. Agr. Res., *40*, 1930, 547–556; McKinney et al., *ibid.*, *26*, 1923, 605–608; Wada and Hukano, Agr. and Hort., *9*, 1934, 1778–1790 (Rev. Appl. Mycol., *14*, 1935, 618, Abst.); Jour. Imp. Agr. Exp. Sta., *3*, 1937, 93–128 (Rev. Appl. Mycol., *16*, 1937, 665, Abst.); Webb, Jour. Agr. Res., *35*, 1927, 587–614; *36*, 1928, 53–75.

41. **Marmor graminis** McKinney. (Jour. Washington Acad. Sci., *34*, 1944, 325.) From Latin *gramen*, grass.

Common name: Brome-grass mosaic virus.

Hosts: *GRAMINEAE—Bromus inermis* Leyss., awnless brome-grass. Experimentally, also *Triticum aestivum* L., wheat; *Avena sativa* L., oat.

Geographical distribution: United States (Kansas).

Induced disease: In awnless bromegrass, systemic chlorotic mottling of the

type commonly called yellow mosaic because of the distinctly yellow color of the chlorotic areas in affected leaves.

Transmission: By inoculation of expressed juice or of aqueous suspensions of dried diseased tissues; not inactivated by drying in diseased tissues for at least 51 days. No insect vector is known.

Literature: McKinney et al., Phytopath., *32*, 1942, 331.

42. **Marmor abaca** H. (*loc. cit.*, 63). From common name of host plant.

Common name: Abacá bunchy-top virus.

Host: *MUSACEAE—Musa textilis* Née, abacá (Manila hemp plant).

Insusceptible species: *MUSACEAE—Musa sapientum* L. vars. *cinerea* (Blanco) Teodoro, *compressa* (Blanco) Teodoro, *lacatan* (Blanco) Teodoro, and *suavcolens* (Blanco) Teodoro; *M. cavendishii* Lamb.

Geographical distribution: Philippine Islands.

Induced disease: In abacá (Manila hemp plant), chlorotic lines and spots along veins of young leaves, followed by growth of distorted leaves, successively shorter, narrower, stiffer, and more curled along their margins. The green areas of mottled leaves, petioles, and leaf sheaths are darker than normal. Newly formed diseased leaves unfurl early, but are short, producing the bunchy top that is referred to in the common name of the disease.

Transmission: By the aphid, *Pentalonia nigronervosa* Coq. (*APHIDIDAE*), vector also of the apparently distinct banana bunchy-top virus of Australia. The incubation period of abacá bunchy-top virus in this aphid is between 24 and 48 hours in length. The progeny of viruliferous aphids do not receive the virus directly, but must feed on diseased plants before they can infect healthy abacá. Transmission by inoculation of expressed juice has not been demonstrated. No soil transmission.

Literature: Ocfemia, Am. Jour. Bot., *17*, 1930, 1–18; Philippine Agriculturist, *22*, 1934, 567–581.

43. **Marmor passiflorae** H. (*loc. cit.*, 77). From New Latin *Passiflora*, generic name of passion fruit.

Common name: Passion-fruit woodiness virus.

Hosts: *PASSIFLORACEAE—Passiflora edulis* Sims, passion fruit; *P. coerulea* L. Experimentally, also *P. alba* Link and Otto.

Insusceptible species: *SOLANACEAE—Datura stramonium* L.; *Lycopersicon esculentum* Mill., tomato; *Nicotiana glutinosa* L.; *N. tabacum* L., tobacco.

Geographical distribution: Australia (New South Wales, Queensland, Victoria), Kenya.

Induced disease: In passion fruit, growth checked; leaves puckered, slightly chlorotic or obscurely mottled, curled, twisted, deformed. Clearing of veins has been observed. Color of stems darker green than normal in some places. Fruits short or deformed, discolored, surface sometimes roughened by cracks; so hard as not to be cut through readily. Pericarp or rind of fruit abnormally thick. Pulp deficient, color deepened. At temperatures below 80° F, some abscission of young chlorotic leaves; above 85° F, masking of the disease in most plants.

Transmission: By inserting cotton in stem wound after soaking it in expressed juice of diseased plant. By aphids, *Myzus persicae* (Sulz.), *Macrosiphum solanifolii* Ashm., and two dark-colored species of the genus *Aphis* (*APHIDIDAE*).

Literature: Cobb, Agr. Gaz. New South Wales, *12*, 1901, 407–418; Noble, Jour. and Proc. Roy. Soc. New South Wales, *62*, 1928, 79–98; Noble and Noble, *ibid.*, *72*, 1939, 293–317; Simmonds, Queensland Agr. Jour., *45*, 1936, 322–330.

44. **Marmor flaccumfaciens** H. (*loc. cit.*, 73). From Latin *flaccus*, flabby, and *facere*, to make.

Common names: Rose-wilt virus, rose dieback virus.

Hosts: *ROSACEAE—Rosa* hybrids, roses.

Geographical distribution: Australia, especially Victoria; New Zealand; possibly Italy.

Induced disease: In rose, leaflets crowded, brittle, recurved. Defoliation progresses from tip to base of plant. Tips of branches discolor and die back an inch or two. Stem darkens at base. Buds remain green and begin development, but growth is soon checked by necrosis at tips. Plant may recover temporarily, but not permanently.

Transmission: By inoculation of expressed juice (needle-puncture and scratch methods). No insect vector is known.

Filterability: Passes Seitz filter (Seitz EK Schichten type, size 6).

Literature: Gigante, Boll. Staz. Pat. Veg. Roma, n. s. *16*, 1936, 76–94; Grieve, Austral. Jour. Exp. Biol. and Med. Science, *8*, 1931, 107–121; Jour. Dept. Agr. Victoria, 1932 and 1933, pages 30–32.

45. **Marmor rosae** H. (*loc. cit.*, 74). From Latin *rosa*, rose.

Common name: Rose-mosaic virus.

Hosts: *ROSACEAE—Rosa rugosa* Thunb.; *R. chinensis* Jacq. var. *manetti* Dipp.; *R. multiflora* Thunb.; *R. odorata* Sweet, tea rose; *R. gymnocarpa*; *Rubus parviflorus* Nutt.

Geographical distribution: United States, England, Bulgaria, Brazil.

Induced disease: In *Rosa rugosa* and *R. chinensis* var. *manetti*, systemic chlorotic mottling.

Transmission: By budding and other forms of graftage. Not by inoculation of expressed juice. No insect vector is known.

Literature: Baker and Thomas, Phytopath., *32*, 1942, 321–326; Brierley, Phytopath., *25*, 1935, 8 (Abst.); Brierley and Smith, Am. Nurseryman, *72*, 1940, 5–8; Jour. Agr. Res., *61*, 1940, 625–660; Kramer, Revista de Agricultura, *15*, 1940, 301–311; O Biologico, *6*, 1940, 365–368; McWhorter, U. S. Dept. Agr., Plant Dis. Rep., *15*, 1931, 1–3; Milbrath, West. Florist, *13*, 1930, 29–30; Nelson, Phytopath., *20*, 1930, 130 (Abst.); Newton, Rep. Domin. Bot., 1930, Div. Bot., Canad. Dept. Agr., 1931, 23; Thomas and Massey, Hilgardia, *12*, 1939, 645–663; Vibert, Jour. Soc. Imp. et Cent. Hort., *9*, 1863, 144–145; White, Phytopath., *22*, 1932, 53–69; *24*, 1934, 1124–1125.

46. **Marmor venenifer**um H. (*loc. cit.*, 75). From Latin *venenifer*, poisonous, in reference to occasional killing of tissues near inserted bud in graft transmission.

Common name: Rose-streak virus.

Hosts: *ROSACEAE—Rosa multiflora* Thunb.; *R. odorata* Sweet; *Rosa* hybrids.

Geographical distribution: Eastern United States.

Induced disease: In various rose species and hybrids, brownish or reddish ring and veinbanding patterns on leaves, and ring patterns on stems. Sometimes necrotic areas near inserted bud, causing girdling of stem and wilting of foliage.

Transmission: By grafting. Not by inoculation of expressed juice. No insect vector is known.

Literature: Brierley, Phytopath., *25*, 1935, 7–8 (Abst.); Brierley and Smith, Jour. Agr. Res., *61*, 1940, 625–660.

47. **Marmor mali** H. (*loc. cit.*, 75). From Latin *malus*, apple tree.

Common name: Apple-mosaic virus.

Hosts: *ROSACEAE—Pyrus malus* L., apple. Experimentally, also *Cotoneaster harroviana*; *Eriobotrya japonica* Lindl., loquat; *Photinia arbutifolia* Lindl., toyon; *Rosa* sp., rose; *Sorbus pallescens*.

Insusceptible species: *ROSACEAE—Amelanchier alnifolia* Nutt.; *Crataegus douglasii* Lindl.; *Pyrus communis* L., pear.

Geographical distribution: United States, Australia, Bulgaria, British Isles.

Induced disease: In apple, clearing of veins and systemic chlorotic spotting. The chlorotic areas sometimes become necrotic during months of intense sunlight.

Transmission: By grafting. No insect

vector is known. Transmission by inoculation of expressed juice has not been demonstrated.

Thermal inactivation: Not demonstrated. Virus in stem tissues withstands at least 50° C for as much as 60 minutes without being inactivated.

Literature: Blodgett, Phytopath., *28*, 1938, 937–938; Bradford and Joley, Jour., Agr. Res., *46*, 1933, 901–908; Christoff, Phytopath. Zeitschr., 7, 1934, 521–536; *8*, 1935, 285–296; Thomas, Hilgardia, *10*, 1937, 581–588.

48. **Marmor fragariae** H. (*loc. cit.*, 78). From New Latin *Fragaria*, generic name of strawberry, from Latin *fraga*, strawberries.

Common name: Strawberry-crinkle virus.

Hosts: *ROSACEAE—Fragaria* hybrids, cultivated strawberries. Experimentally, also *Fragaria vesca* L., woodland strawberry.

Geographical distribution: United States, England.

Induced disease: In cultivated strawberry, crinkling and chlorosis of leaves. At first, minute chlorotic flecks appear in young leaves. These flecks enlarge, and small necrotic spots may appear in their centers. Vein-clearing appears frequently. Affected foliage lighter and less uniformly green than normal. The variety Royal Sovereign may appear normal through carrying this virus.

Transmission: By aphid, *Myzus fragaefolii* Ckll. (= *Capitophorus fragariae* Theob.) (*APHIDIDAE*). By grafting. Not by inoculation of expressed juice.

Literature: Harris, Ann. Rept. East Malling Res. Sta. for 1936, 1937, 201–211, 212–221; *ibid.*, for 1937, 1938, 201–202; Harris and Hildebrand, Canad. Jour. Res., C, *15*, 1937, 252–280; Ogilvie et al., Ann. Rept. Long Ashton Res. Sta. for 1933, 1934, 96–97; Vaughan, Phytopath., *23*, 1933, 738–740; Zeller, Oregon Agr. Exp. Sta., Sta. Bull. 319, 1933; Zeller and Vaughan, Phytopath., *22*, 1932, 709–713.

49. **Marmor marginans** H. (*loc. cit.*, 79). From Latin *marginare*, to provide with a margin.

Common name: Strawberry yellow-edge virus.

Hosts: *ROSACEAE—Fragaria* hybrids, strawberries; *Fragaria californica* C. and S.; *F. chiloensis* Duch. (symptomless). Experimentally, also *Fragaria vesca* L.; *F. virginiana* Duch. (some clones appear to be immune to infection by runner inarching).

Geographical distribution: United States, England, France, New Zealand.

Induced disease: In strawberry, plant appears flat with outer zone of leaves more or less normal, central leaves dwarfed, yellow-edged, deficient in red pigmentation. The variety Premier may carry this virus without showing any obvious manifestation of disease.

Transmission: By aphid, *Myzus fragaefolii* Ckll. (*APHIDIDAE*). By grafting. Not by inoculation of expressed juice. Not through seeds from diseased plants.

Literature: Chamberlain, New Zealand Jour. Agr., *49*, 1934, 226–231; Harris, Jour. Pom. and Hort. Science, *11*, 1933, 56–76; Harris and Hildebrand, Canad. Jour. Res., C, *15*, 1937, 252–280; Hildebrand, *ibid.*, C, *19*, 1941, 225–233; Plakidas, Phytopath., *16*, 1926, 423–426; Jour. Agr. Res., *35*, 1927, 1057–1090.

50. **Marmor rubi** H. (Holmes, *loc. cit.*, 80; *Poecile rubi* McKinney, Jour. Washington Acad. Science, *34*, 1944, 148.) From Latin *rubus*, bramble bush.

Common name: Red-raspberry mosaic virus.

Hosts: *ROSACEAE—Rubus idaeus* L., red raspberry; *R. occidentalis* L., black raspberry.

Geographical distribution: United States.

Induced disease: In red raspberry, systemic chlorotic mottling, masked at high temperatures of summer. Foliage development delayed in spring. In some varieties, leaf petioles and cane tips die,

canes remain short and become rosetted.

Transmission: By aphids, principally *Amphorophora rubi* Kalt., but also *A. rubicola* Oestl. and *A. sensoriata* Mason (*APHIDIDAE*). Not by inoculation of expressed juice.

Literature: Bennett, Michigan Agr. Exp. Sta., Techn. Bull. 80, 1927; 125, 1932; Cooley, New York Agr. Exp. Sta. (Geneva), Bull. 675, 1936; Harris, Jour. Pom. and Hort. Science, *11*, 1933, 237–255; *17*, 1940, 318–343; Rankin, New York Agr. Exp. Sta., Geneva, Bull. 543, 1927; New York Agr. Exp. Sta., Geneva, Tech. Bull. 175, 1931.

51. **Marmor persicae** H. (Holmes, *loc. cit.*, 81; *Flavimacula persicae* McKinney, Jour. Washington Acad. Science, *34*, 1944, 149.) From New Latin *Persica*, former generic name of peach.

Common name: Peach-mosaic virus.

Hosts: *ROSACEAE—Prunus persica* (L.) Batsch, peach and nectarine, all tested varieties. Experimentally, also *P. armeniaca* L., apricot; *P. communis* Fritsch, almond; *P. domestica* L., plum and prune.

Insusceptible species: Attempts to infect sweet and sour cherries have thus far failed.

Geographical distribution: United States (Colorado, California, Utah, Oklahoma, Texas, New Mexico, Arizona).

Induced disease: In peach, short internodes in spring growth, sometimes breaking in flower pattern, chlorotic mottling and distortion of foliage early in season, masking of leaf symptoms or excision of affected areas of leaf lamina in midsummer; fruit small, irregular in shape, unsalable. Some peach varieties are less damaged than others, but all are thought to be equally susceptible to infection, and equally important as reservoirs of virus when infected. In almond, experimentally, symptomless infections; symptoms appear in some apricot and plum varieties when experimentally infected, not in others.

Transmission: By budding and other methods of grafting. Not by inoculation of expressed juice. Not through soil. No insect vector is known. Not through pollen or seed from diseased plants.

Thermal inactivation: Not demonstrated; virus not inactivated by temperatures effective in inactivating peach-yellows virus.

Literature: Bodine, Colorado Agr. Exp. Sta., Bull. 421, 1936; Bodine and Durrell, Phytopath., *31*, 1941, 322–333; Cation, *ibid.*, 24, 1934, 1380–1381; Christoff, Phytopath. Ztschr., *11*, 1938, 360–422; Cochran, California Cultivator, *87*, 1940, 164–165; Cochran and Hutchins, Phytopath., *28*, 1938, 890–892; Hutchins, Science, *76*, 1932, 123; Hutchins et al., U. S. Dept. Agr., Circ. 427, 1937, 48 pp.; Kunkel, Am. Jour. Bot., *23*, 1936, 683–686; Phytopath., *28*, 1938, 491–497; Thomas and Rawlins, Hilgardia, *12*, 1939, 623–644; Valleau, Kentucky Agr. Exp. Sta., Bull. 327, 1932, 89–103.

52. **Marmor astri** H. (*loc. cit.*, 83). From Latin *astrum*, star.

Common name: Peach asteroid-spot virus.

Host: *ROSACEAE—Prunus persica* (L.) Batsch, peach.

Geographical distribution: California.

Induced disease: In peach, discrete chlorotic lesions spreading along veins, forming star-like spots; developing leaves normal in appearance, becoming affected as they mature. Some chlorophyll retained in lesions as leaves turn yellow. Affected leaves shed early.

Transmission: By grafting. Not by inoculation of expressed juice. No insect vector is known.

Literature: Cochran and Smith, Phytopath., *28*, 1938, 278–281.

53. **Marmor rubiginosum** Reeves. (Phytopath., *30*, 1940, 789.) From Latin *rubiginosus*, rusty.

Common name: Cherry rusty-mottle virus.

Host: *ROSACEAE—Prunus avium* L., sweet cherry.

Geographical distribution: United States (Washington).

Induced disease: In sweet cherry, chlorotic mottling 4 to 5 weeks after full bloom, first on small basal leaves, later on all leaves. The older affected leaves develop autumnal colors and absciss, 30 to 70 per cent of the foliage being lost. The remaining foliage appears somewhat wilted, shows increased mottling, chlorotic spots, and areas becoming yellowish brown, appearing rusty. Blossoms normal. Fruits smaller than normal, insipid, not misshapen. Growth rate of tree reduced slightly.

Transmission: By grafting. Not by inoculation of expressed juice. No insect vector is known.

Literature: Reeves, Phytopath., *30*, 1940, 789 (Abst.); Jour. Agr. Res., *62*, 1941, 555–572 (see 566–567).

54. **Marmor cerasi** Zeller and Evans. (Phytopath., *31*, 1941, 467.) From Latin *cerasus*, cherry tree; originally spelled *cerasae*, by error.

Common name: Cherry mottle-leaf virus.

Hosts: *ROSACEAE—Prunus avium* L., sweet cherry; *P. emarginata* (Dougl.) Walp., wild cherry. Experimentally, also *P. cerasus* L. (tolerant) and *P. mahaleb* L. (tolerant).

Geographical distribution: United States (Washington, Oregon, Idaho, California) and Canada (British Columbia).

Induced disease: In sweet cherry, chlorotic mottling; leaves puckered, wrinkled, distorted, not perforated. Blossoms not affected. Fruit small, hard, insipid, uneven or delayed in ripening. Crop reduced. Branches shortened, tree eventually stunted.

Transmission: By budding. No insect vector is known. Not by the black cherry aphid, *Myzus cerasi* (F.) (*APHIDIDAE*). Not by inoculation of expressed juice.

Thermal inactivation: Not demonstrated; not at 46° C in 60 minutes nor at 49° C in 10 minutes in bud sticks.

Literature: Reeves, Washington State Hort. Assoc. Proc., *31*, 1935, 85–89; Jour. Agr. Res., *62*, 1941, 555–572; Zeller, Oregon State Hort. Soc. Report, 26, 1934, 92–95; Phytopath., *31*, 1941, 463–467.

55. **Marmor lineopictum** Cation (Phytopath., *31*, 1941, 1009.) From Latin *linea*, line, and *pictus*, ornamented.

Common names: Prunus line-pattern virus, peach line-pattern virus.

Hosts: *ROSACEAE—Prunus salicina* Lindl., Japanese plum; *P. mahaleb* L., Mahaleb cherry; *P. persica* (L.) Batsch, peach (= *Amygdalus persica* L.).

Geographical distribution: United States (Kentucky, Michigan, California, Ohio; perhaps widely distributed).

Induced disease: In peach and Mahaleb cherry, light-colored line patterns or faint chlorotic mottling, tending to become masked as leaf becomes old. In peach, affected foliage sometimes less glossy than normal. In *Prunus salicina*, no disease manifestations usually; rarely, chlorotic mottling on a few leaves.

Transmission: By grafting. No insect vector is known.

Literature: Berkeley, Div. of Bot. and Plant Path., Science Service, Dominion Dept. Agr., Ottawa, Canada, Publ. 679, 1941; Cation, Phytopath., *31*, 1941, 1004–1010; Thomas and Rawlins, Hilgardia, *12*, 1939, 623–644; Valleau, Kentucky Agr. Exp. Sta., Res. Bull. 327, 1932, 89–103.

56. **Marmor pallidolimbatus** Zeller and Milbrath. (In Handbook of Virus Diseases of Stone Fruits in North America, Michigan Agr. Exp. Sta., Miscell. Publ., May, 1942, 50; Phytopath., *32*, 1942, 635.) From Latin *pallidus*, pale, and *limbatus*, bordered.

Common name: Cherry banded-chlorosis virus.

Hosts: *ROSACEAE—Prunus serrulata* Lindl., flowering cherry; *P. avium* L., Mazzard cherry.

Geographical distribution: United States (Pacific Northwest).

Induced disease: In flowering cherry,

canes remain short and become rosetted.

Transmission: By aphids, principally *Amphorophora rubi* Kalt., but also *A. rubicola* Oestl. and *A. sensoriata* Mason (*APHIDIDAE*). Not by inoculation of expressed juice.

Literature: Bennett, Michigan Agr. Exp. Sta., Techn. Bull. 80, 1927; 125, 1932; Cooley, New York Agr. Exp. Sta. (Geneva), Bull. 675, 1936; Harris, Jour. Pom. and Hort. Science, *11*, 1933, 237–255; *17*, 1940, 318–343; Rankin, New York Agr. Exp. Sta., Geneva, Bull. 543, 1927; New York Agr. Exp. Sta., Geneva, Tech. Bull. 175, 1931.

51. **Marmor persicae** H. (Holmes, *loc. cit.*, 81; *Flavimacula persicae* McKinney, Jour. Washington Acad. Science, *34*, 1944, 149.) From New Latin *Persica*, former generic name of peach.

Common name: Peach-mosaic virus.

Hosts: *ROSACEAE—Prunus persica* (L.) Batsch, peach and nectarine, all tested varieties. Experimentally, also *P. armeniaca* L., apricot; *P. communis* Fritsch, almond; *P. domestica* L., plum and prune.

Insusceptible species: Attempts to infect sweet and sour cherries have thus far failed.

Geographical distribution: United States (Colorado, California, Utah, Oklahoma, Texas, New Mexico, Arizona).

Induced disease: In peach, short internodes in spring growth, sometimes breaking in flower pattern, chlorotic mottling and distortion of foliage early in season, masking of leaf symptoms or excision of affected areas of leaf lamina in midsummer; fruit small, irregular in shape, unsalable. Some peach varieties are less damaged than others, but all are thought to be equally susceptible to infection, and equally important as reservoirs of virus when infected. In almond, experimentally, symptomless infections; symptoms appear in some apricot and plum varieties when experimentally infected, not in others.

Transmission: By budding and other methods of grafting. Not by inoculation of expressed juice. Not through soil. No insect vector is known. Not through pollen or seed from diseased plants.

Thermal inactivation: Not demonstrated; virus not inactivated by temperatures effective in inactivating peach-yellows virus.

Literature: Bodine, Colorado Agr. Exp. Sta., Bull. 421, 1936; Bodine and Durrell, Phytopath., *31*, 1941, 322–333; Cation, *ibid.*, 24, 1934, 1380–1381; Christoff, Phytopath. Ztschr., *11*, 1938, 360–422; Cochran, California Cultivator, *87*, 1940, 164–165; Cochran and Hutchins, Phytopath., *28*, 1938, 890–892; Hutchins, Science, *76*, 1932, 123; Hutchins et al., U. S. Dept. Agr., Circ. 427, 1937, 48 pp.; Kunkel, Am. Jour. Bot., *23*, 1936, 683–686; Phytopath., *28*, 1938, 491–497; Thomas and Rawlins, Hilgardia, *12*, 1939, 623–644; Valleau, Kentucky Agr. Exp. Sta., Bull. 327, 1932, 89–103.

52. **Marmor astri** H. (*loc. cit.*, 83). From Latin *astrum*, star.

Common name: Peach asteroid-spot virus.

Host: *ROSACEAE—Prunus persica* (L.) Batsch, peach.

Geographical distribution: California.

Induced disease: In peach, discrete chlorotic lesions spreading along veins, forming star-like spots; developing leaves normal in appearance, becoming affected as they mature. Some chlorophyll retained in lesions as leaves turn yellow. Affected leaves shed early.

Transmission: By grafting. Not by inoculation of expressed juice. No insect vector is known.

Literature: Cochran and Smith, Phytopath., *28*, 1938, 278–281.

53. **Marmor rubiginosum** Reeves. (Phytopath., *30*, 1940, 789.) From Latin *rubiginosus*, rusty.

Common name: Cherry rusty-mottle virus.

Host: *ROSACEAE—Prunus avium* L., sweet cherry.

Geographical distribution: United States (Washington).

Induced disease: In sweet cherry, chlorotic mottling 4 to 5 weeks after full bloom, first on small basal leaves, later on all leaves. The older affected leaves develop autumnal colors and abscise, 30 to 70 per cent of the foliage being lost. The remaining foliage appears somewhat wilted, shows increased mottling, chlorotic spots, and areas becoming yellowish brown, appearing rusty. Blossoms normal. Fruits smaller than normal, insipid, not misshapen. Growth rate of tree reduced slightly.

Transmission: By grafting. Not by inoculation of expressed juice. No insect vector is known.

Literature: Reeves, Phytopath., *30*, 1940, 789 (Abst.); Jour. Agr. Res., *62*, 1941, 555–572 (see 566–567).

54. **Marmor cerasi** Zeller and Evans. (Phytopath., *31*, 1941, 467.) From Latin *cerasus*, cherry tree; originally spelled *cerasae*, by error.

Common name: Cherry mottle-leaf virus.

Hosts: *ROSACEAE—Prunus avium* L., sweet cherry; *P. emarginata* (Dougl.) Walp., wild cherry. Experimentally, also *P. cerasus* L. (tolerant) and *P. mahaleb* L. (tolerant).

Geographical distribution: United States (Washington, Oregon, Idaho, California) and Canada (British Columbia).

Induced disease: In sweet cherry, chlorotic mottling; leaves puckered, wrinkled, distorted, not perforated. Blossoms not affected. Fruit small, hard, insipid, uneven or delayed in ripening. Crop reduced. Branches shortened, tree eventually stunted.

Transmission: By budding. No insect vector is known. Not by the black cherry aphid, *Myzus cerasi* (F.) (*APHIDIDAE*). Not by inoculation of expressed juice.

Thermal inactivation: Not demonstrated; not at 46° C in 60 minutes nor at 49° C in 10 minutes in bud sticks.

Literature: Reeves, Washington State Hort. Assoc. Proc., *31*, 1935, 85–89; Jour. Agr. Res., *62*, 1941, 555–572; Zeller, Oregon State Hort. Soc. Report, 26, 1934, 92–95; Phytopath., *31*, 1941, 463–467.

55. **Marmor lineopictum** Cation (Phytopath., *31*, 1941, 1009.) From Latin *linea*, line, and *pictus*, ornamented.

Common names: Prunus line-pattern virus, peach line-pattern virus.

Hosts: *ROSACEAE—Prunus salicina* Lindl., Japanese plum; *P. mahaleb* L., Mahaleb cherry; *P. persica* (L.) Batsch, peach (= *Amygdalus persica* L.).

Geographical distribution: United States (Kentucky, Michigan, California, Ohio; perhaps widely distributed).

Induced disease: In peach and Mahaleb cherry, light-colored line patterns or faint chlorotic mottling, tending to become masked as leaf becomes old. In peach, affected foliage sometimes less glossy than normal. In *Prunus salicina*, no disease manifestations usually; rarely, chlorotic mottling on a few leaves.

Transmission: By grafting. No insect vector is known.

Literature: Berkeley, Div. of Bot. and Plant Path., Science Service, Dominion Dept. Agr., Ottawa, Canada, Publ. 679, 1941; Cation, Phytopath., *31*, 1941, 1004–1010; Thomas and Rawlins, Hilgardia, *12*, 1939, 623–644; Valleau, Kentucky Agr. Exp. Sta., Res. Bull. 327, 1932, 89–103.

56. **Marmor pallidolimbatus** Zeller and Milbrath. (In Handbook of Virus Diseases of Stone Fruits in North America, Michigan Agr. Exp. Sta., Miscell. Publ., May, 1942, 50; Phytopath., *32*, 1942, 635.) From Latin *pallidus*, pale, and *limbatus*, bordered.

Common name: Cherry banded-chlorosis virus.

Hosts: *ROSACEAE—Prunus serrulata* Lindl., flowering cherry; *P. avium* L., Mazzard cherry.

Geographical distribution: United States (Pacific Northwest).

Induced disease: In flowering cherry,

chlorotic bands surrounding discolored areas on leaves. In Mazzard cherry, dwarfing of whole plant, chlorotic bands on leaves.

Transmission: By budding, even in the absence of survival of inserted buds.

57. **Marmor nerviclarens** Zeller and Evans. (Phytopath., *31*, 1941, 467.) From Latin *nervus*, sinew or nerve, and *clarere*, to shine.

Common name: Cherry vein-clearing virus.

Hosts: *ROSACEAE—Prunus avium* L., sweet cherry. Perhaps also *P. serrulata* Lindl. and *P. domestica* L., on which symptoms similar to those induced by this virus have been observed.

Geographical distribution: United States (Oregon, Washington).

Induced disease: In sweet cherry, clearing of veins throughout each leaf or only in localized areas. Margins of leaves irregular, most indented where clearing of veins is most conspicuous. Elongated, elliptic, or slot-like perforations occur in some leaves. Affected leaves usually narrow. Enations occur as small blistered proliferations on lower side of main veins. Upper leaf surface silvery by reflected light. By midsummer, leaves droop and appear somewhat wilted; they may fold along the midvein. Internodes short; increased number of buds, spurs, or short branches at nodes; rosetting more pronounced on some branches than on others, mostly at end of year-old wood. In advanced disease, fruits pointed, small, flattened on suture side with swollen ridge along suture. Blossoms abnormally abundant, crop of fruit reduced or wanting.

Transmission: By grafting. Not demonstrated by inoculation of expressed juice. No insect vector is known.

58. **Marmor viticola** H. (*loc. cit.*, 83). From Latin *vitis*, vine, and *-cola*, inhabitant of.

Common name: Vine-mosaic virus.

Host: *VITACEAE—Vitis vinifera* L., grape.

Geographical distribution: France, Italy, Bulgaria, Czechoslovakia.

Induced disease: In grape, various modifications of systemic chlorotic mottling, and red pigmentation of parts of leaves with subsequent drying and dropping out of affected spots. Leaves deformed, crimped between main veins. Growth restricted.

Transmission: By inoculation of expressed juice and by pruning.

Literature: Blattný, Vinařský obzor., *25*, 1931, 4–5 (Cent. f. Bakt., II Abt., *84*, 1931, 464); Ochrana Rostlin, *13*, 1933, 104–105 (Rev. Appl. Mycol., *13*, 1934, 421); Gigante, Boll. Staz. Pat. Veg. Roma, n. s. *17*, 1937, 169–192 (Rev. Appl. Mycol., *17*, 1938, 221); Pantanelli, Malpighia, *24*, 1911, 497–523; *25*, 1912, 17–46; Stranak, II Congr. Intern. Path. Comp. Paris, 1931, 367–378; Ochrana Rostlin, *11*, 1931, 89–98 (Rev. Appl. Mycol., *11*, 1932, 280); Vielwerth, Ochrana Rostlin, *13*, 1933, 83–90 (Rev. Appl. Mycol., *13*, 1934, 421–422).

59. **Marmor santali** H. (*loc. cit.*, 94). From New Latin, *Santalum*, generic name of sandal.

Common name: Sandal leaf-curl virus.

Host: *SANTALACEAE—Santalum album* L., sandal.

Geographical distribution: India.

Induced disease: In sandal, leaves small, curled, wrinkled, thickened, brittle, abscissing. Systemic chlorotic mottling. Internode length normal. Infected twigs produce both flowers and fruits.

Transmission: By ring bark-grafts. Not by inoculation of expressed juice. No insect vector is known.

Literature: Venkata Rao, Mysore Sandal Spike Invest. Comm., Bull. 3, 1933.

60. **Marmor secretum** Bennett. (Phytopath., *34*, 1944, 88). From Latin *secretus*, hidden.

Common name: Dodder latent-mosaic virus.

Hosts: *CONVOLVULACEAE—Cuscuta californica* Choisy, dodder. Experi-

mentally, also *CHENOPODIACEAE—Beta vulgaris* L., sugar beet; *Chenopodium album* L., lamb's quarters; *C. murale* L., sowbane. *CONVOLVULACEAE—Cuscuta campestris* Yuncker; *C. subinclusa* Dur. and Hilg. *CRUCIFERAE—Brassica incana* (L.) F. W. Schultz, mustard (tolerant). *CUCURBITACEAE—Cucumis melo* L., cantaloupe. *PHYTOLACCACEAE—Phytolacca americana* L., pokeweed. *PLANTAGINACEAE—Plantago major* L., plantain. *POLYGONACEAE—Fagopyrum esculentum* Moench, buckwheat; *Polygonum pennsylvanicum* L., knotweed. *PRIMULACEAE—Samolus floribundus* HBK., water pimpernel. *SOLANACEAE—Lycopersicon esculentum* Mill., tomato; *Nicotiana glauca* Graham (tolerant); *N. palmeri* Gray; *N. rustica* L. (tolerant); *N. tabacum* L. (tolerant); *Solanum tuberosum* L., potato. *UMBELLIFERAE—Apium graveolens* L., celery.

Insusceptible species: *COMPOSITAE—Helianthus annuus* L., sunflower; *Lactuca sativa* L., lettuce. *CRUCIFERAE—Brassica oleracea* L., cabbage. *POLYGONACEAE—Eriogonum fasciculatum* Benth., California buckwheat. *SCROPHULARIACEAE—Verbascum thapsus* L., mullein. *SOLANACEAE—Atropa belladonna* L., belladonna.

Geographical distribution: United States (California).

Induced disease: In dodder, no symptoms. In sugar beet, experimentally, temporary systemic chlorotic spotting; occasional faded areas in leaves in subsequent chronic stage of disease. In cantaloupe, experimentally, chlorotic spotting, reduction in leaf size, death of some leaves, stunting of plant; melons small and of poor quality. In celery, experimentally, systemic chlorosis followed by dwarfing and mottling with subsequent apparent recovery.

Transmission: By dodder, *Cuscuta californica, C. campestris,* and *C. subinclusa.* By inoculation of extracted juice to some, but not to other, host plants; *Phytolacca americana* is readily infected by rubbing methods in the presence of a small amount of abrasive, and develops numerous necrotic primary lesions that serve for quantitative estimation of concentration of virus in inoculum. Through seeds from infected plants of dodder, *Cuscuta campestris*; not through seeds from diseased cantaloupe, buckwheat, or pokeweed plants. No insect vector is known.

Thermal inactivation: At 56 to 60° C in 10 minutes.

Filterability: Passes celite and Berkefeld N and W filters.

Other properties: Infective in dilutions to 1:3000. Inactivated by drying and by storage in expressed pokeweed juice, within 48 hours.

61. **Marmor pelargonii** *spec. nov.* From New Latin *Pelargonium,* generic name of common geranium.

Common names: Pelargonium leaf-curl virus; virus of dropsy or Kräuselkrankheit of geranium.

Host: *GERANIACEAE—Pelargonium hortorum* Bailey, geranium.

Induced disease: In geranium, circular or irregular chlorotic spots, sometimes stellate or dendritic, ½ to 5 mm in diameter, centers becoming brown with chlorotic border; severely affected leaves become yellow and drop; spotted leaves ruffled, crinkled, malformed, small, sometimes puckered and splitting. Petioles and stems show corky, raised, necrotic streaks; tops may die. Disease most severe in spring, inconspicuous in summer.

Transmission: By grafting. Not by inoculation of expressed juice nor by use of knife to prepare cuttings for propagation. Not through seed. No insect vector is known.

Literature: Berkeley, Canad. Hort. and Home Mag., *1938,* 1938, 1–4; Blattný, Ochrana Rostlin, *13,* 1933, 145 (Rev. Appl. Mycol., *13,* 1934, 378–379); Bremer, Blumen-u. Pflanzenbau, *48,* 1933, 32–33 (Rev. Appl. Mycol., *12,* 1933, 514); Halstead, New Jersey Agr. Exp. Sta., Rept. 14, 1893, 432–433; Jones, Washington Agr. Exp. Sta., Bull. 390, 1940;

Laubert, Gartenwelt, *31*, 1927, 391; Pape, *ibid.*, *26*, 1927, 329–331; *32*, 1928, 116–117; Pethybridge and Smith, Gard. Chron., *92*, 1932, 378–379; Schmidt, Gartenwelt, *31*, 1932, 40; Seeliger, Nachrichtenbl. Deutsch. Pflanzenschutzdienst, *6*, 1926, 63–64; Tuimann, Gartenwelt, *31*, 1927, 375–376; Verplancke, Bul. Cl. Sci. Acad. Roy. de Belgique, Ser. 5, *18*, 1932, 269–281 (Rev. Appl. Mycol., *11*, 1932, 649–650).

62. **Marmor angliae** H. (*loc. cit.*, 48). From Latin *Anglia*, England.

Common name: Potato-paracrinkle virus.

Hosts: *SOLANACEAE—Solanum tuberosum* L., potato. Experimentally, also *Datura stramonium* L., Jimson weed.

Insusceptible species: *SOLANACEAE—Nicotiana tabacum* L., tobacco.

Geographical distribution: England.

Induced disease: In potato, masked in all plants of the variety King Edward. Chlorotic mottling with some necrosis in the varieties Arran Victory and Arran Chief. Chlorotic mottling only in Arran Comrade, Majestic, and Great Scot potatoes. Two varieties, Sharpe's Express and Epicure, are said to be resistant.

Transmission: By grafts. Not by inoculation of expressed juice. No insect vector is known.

Literature: Dykstra, Phytopath., *26*, 1936, 597–606; Salaman and Le Pelley, Proc. Roy. Soc. London, Ser. B, *106*, 1930, 140–175.

63. **Marmor aevi** *spec. nov.* From Latin *aevum*, old age, in reference to the obvious involvement of old, but not of young, delphinium leaves.

Common name: Celery-calico virus.

Hosts: *CUCURBITACEAE—Cucumis sativus* L., cucumber; *C. melo* L., cantaloupe; *Cucurbita pepo* L., summer crookneck squash. *RANUNCULACEAE—Delphinium chinensis*; *D. formosum*, hardy larkspur; *D. grandiflorum; D. parryi; D. zalil. SOLANACEAE—Lycopersicon esculentum* Mill., tomato. *UMBELLIFERAE—Apium graveolens* L., celery. Experimentally, also *SOLANACEAE—Nicotiana tabacum* L., tobacco; *Petunia hybrida* Vilm., petunia. *VIOLACEAE—Viola cornuta* L.

Geographical distribution: United States.

Induced disease: In celery, clearing of veins, puckering and downward cupping of younger leaves, green islands of tissue in lemon-yellow areas of outer leaves, green and yellow zigzag bands on leaflets. In delphinium, basal and middle leaves with pale-orange, amber, or lemon-yellow areas; younger leaves normal green; chlorotic ring and line patterns.

Transmission: By inoculation of expressed juice in the presence of finely powdered carborundum. By aphids: *Aphis apigraveolens* Essig, celery leaf aphid; *A. apii* Theob., celery aphid; *A. ferruginea-striata* Essig, rusty-banded aphid; *A. gossypii* Glov., cotton aphid; *A. middletonii* Thomas, erigeron root aphid; *Myzus circumflexus* (Buckt.), lily aphid; *M. convolvuli* (Kalt.), foxglove aphid; *M. persicae* (Sulz.), green peach aphid; *Rhopalosiphum melliferum* (Hottes), honeysuckle aphid (*APHIDIDAE*).

Literature: Severin, Hilgardia, *14*, 1942, 441–464; Severin and Freitag, Phytopath., *25*, 1935, 891 (Abst.); Hilgardia, *11*, 1938, 493–558.

64. **Marmor raphani** *spec. nov.* From Latin *raphanus*, radish.

Common name: Radish-mosaic virus.

Hosts: *CRUCIFERAE—Raphanus sativus* L., radish. Experimentally, also *CRUCIFERAE—Brassica oleracea* L.; *B. nigra* (L.) Koch; *B. alba* (L.) Boiss; *B. arvensis* (L.) Ktze.; *B. pe-tsai* Bailey; *B. juncea* (L.) Coss; *B. rapa* L.; *B. adpressa* Boiss; *Capsella bursa-pastoris* (L.) Medic.; *Malcomia maritima* R. Br.; *M. bicornis* DC. *CHENOPODIACEAE—Chenopodium album* L.; *C. murale* L.; *Spinacia oleracea* L. *RANUNCULACEAE—Delphinium ajacis* L. *SOLANACEAE—Nicotiana glutinosa* L.; *N.*

langsdorffii Weinm.; *N. rustica* L.; *N. tabacum* L.

Geographical distribution: United States (California).

Induced disease: In radish, systemic chlorotic spotting followed by chlorotic mottling of foliage; little or no leaf distortion; plants not stunted.

Transmission: By inoculation of expressed juice. No insect vector is known; not by the cabbage aphid, *Brevicoryne brassicae* (L.); the false cabbage aphid, *Lipaphis pseudobrassicae* (Davis); or the green peach aphid, *Myzus persicae* (Sulz.) (*APHIDIDAE*). Not through seeds from diseased radish plants.

Thermal inactivation: At 65 to 68° C in 10 minutes.

Literature: Tompkins, Jour. Agr. Res., *58*, 1939, 119–130.

65. **Marmor primulae** *spec. nov.* From New Latin *Primula*, generic name of primrose.

Common name: Primrose-mosaic virus.

Hosts: *PRIMULACEAE—Primula obconica* Hance. Experimentally, also *P. malacoides* Franch. and *P. sinensis* Lindl.

Insusceptible species: *BEGONIACEAE—Begonia semperflorens* Link and Otto. *BORAGINACEAE—Myosotis alpestris* Schmidt. *CAMPANULACEAE—Campanula medium* L. *CARYOPHYLLACEAE—Dianthus barbatus* L. *CHENOPODIACEAE—Spinacia oleracea* L. *COMPOSITAE—Bellis perennis* L.; *Callistephus chinensis* Nees; *Gerbera jamesonii* Hook.; *Lactuca sativa* L.; *Senecio cruentus* DC.; *Tagetes patula* L. *CRUCIFERAE—Brassica oleracea* L.; *B. pe-tsai* Bailey; *B. rapa* L.; *Matthiola incana* R. Br.; *Raphanus sativus* L. *CUCURBITACEAE—Cucumis sativus* L.; *Cucurbita pepo* L. *EUPHORBIACEAE—Ricinus communis* L. *GRAMINEAE—Zea mays* L. *LEGUMINOSAE—Pisum sativum* L.; *Vicia faba* L.; *Vigna sinensis* (Torner) Savi. *LOBELIACEAE—Lobelia hybrida* Hort. *PAPAVERACEAE—Papaver orientale* L. *PRIMULACEAE—Anagallis arvensis* L.; *Cyclamen indicum* L.; *Primula auricula* L.; *P. veris* L. *RANUNCULACEAE—Anemone coronaria* L.; *Delphinium cultorum* Voss; *Ranunculus asiaticus* L. *RESEDACEAE—Reseda odorata* L. *ROSACEAE—Geum chiloense* Balb. *SCROPHULARIACEAE—Antirrhinum majus* L.; *Pentstemon barbatus* Nutt. *SOLANACEAE—Capsicum frutescens* L.; *Datura stramonium* L.; *Lycopersicon esculentum* Mill.; *Nicotiana glutinosa* L.; *N. tabacum* L.; *Solanum tuberosum* L. *TROPAEOLACEAE—Tropaeolum majus* L. *UMBELLIFERAE—Apium graveolens* L. *VERBENACEAE—Verbena hybrida* Voss. *VIOLACEAE—Viola tricolor* L.

Geographical distribution: United States (California).

Induced disease: In *Primula obconica*, chlorosis, stunting, rugosity with upward, or occasionally downward, cupping of leaves. Petioles and peduncles shortened; flowers reduced in size, broken in color (white-streaked). Leaves coarsely mottled with yellow-green, leaving green islands; tips of leaves sometimes narrowed.

Transmission: By inoculation of expressed juice, in the presence of 600-mesh powdered carborundum. Not by aphids, *Myzus persicae* (Sulz.) and *M. circumflexus* (Buckt.) (*APHIDIDAE*). No insect vector is known. Probably not through seeds.

Thermal inactivation: At 50° C, not 48° C, in 10 minutes.

Other properties: Infective after 24, not 48, hours *in vitro*. Infective after 1:10 dilution.

Literature: Tompkins and Middleton, Jour. Agr. Res., *63*, 1941, 671–679.

66. **Marmor caricae** (Condit and Horne) *comb. nov.* (*Ficivir caricae* Condit and Horne, Phytopath., *31*, 1941, 563.) From Latin *carica*, a kind of dried fig.

Common name: Fig-mosaic virus.

Hosts: *MORACEAE—Ficus carica* L.,

fig; *F. altissima* Blume; *F. krishna*; and *F. tsiela* Roxb.

Geographical distribution: United States (California, Texas), England, Puerto Rico, China, New South Wales, Western Australia.

Induced disease: In fig, systemic chlorotic spotting and mottling of foliage; some severe leaf distortion. Fruits sometimes affected, bearing light circular areas, rusty spots, being deformed or dropped prematurely. Necrotic lesions on profichi of Samson caprifigs also have been attributed to action of this virus.

Transmission: By budding. No insect vector is known; mites have been suspected as possible vectors.

Literature: Condit and Horne, Phytopath., *23*, 1933, 887–896; *31*, 1941, 561–563; *33*, 1943, 719–723; Ho and Li, Lingnan Science Jour., *15*, 1936, 69–70; Pittman, W. Austral. Dept. Agr. Jour., *12*, 1935 196.

67. **Marmor italicum** (Fawcett) *comb. nov.* (*Citrivir italicum* Fawcett, Phytopath., *31*, 1941, 357.) Specific epithet meaning "pertaining to Italy."

Common name: Citrus infectious-mottling virus.

Host: *RUTACEAE—Citrus aurantium* L., sour orange.

Geographical distribution: Italy.

Induced disease: In sour orange, white, pale green, or yellow irregular areas in leaves, leaving narrow green bands along midrib; leaves blistered and distorted.

Transmission: The aphid, *Toxoptera aurantii* (Phytopath., *24*, 1934, 661), has been suspected as vector.

Literature: Fawcett, Phytopath., *31*, 1941, 356–357; Petri, Bol. Staz. Pat. Veg. Roma, n. s. *11*, 1931, 105–114.

NOTE: Several additional species were described too late for complete systematic treatment here. They are plain's wheat mosaic virus, **Marmor campestre** McKinney (Jour. Washington Acad. Sci., *34*, 1944, 324) with varieties *typicum* McKinney and *galbinum* McKinney, respectively causing light-green mosaic and severe yellow mosaic of wheat in Kansas; wheat streak-mosaic virus, **Marmor virgatum** McKinney (*ibid.*, *34*, 1944, 324) with varieties *typicum* McKinney and *viride* McKinney (*ibid.*, *34*, 1944, 325), respectively causing yellow streak-mosaic and green streak-mosaic of wheat in Kansas; Agropyron-mosaic virus, **Marmor agropyri** McKinney (*ibid.*, *34*, 1944, 326), with varieties *typicum* McKinney and *flavum* McKinney, respectively causing green-mosaic mottling and yellow-mosaic mottling in the grass *Agropyron repens* (L.) Beauv. in Virginia; also a virus, **Flavimacula ipomeae** Doolittle and Harter (Phytopath., *35*, 1945, 703), causing feathery mottle of sweet potato in Maryland [see *Marmor persicae* for treatment of a virus that was assigned as type of *Flavimacula* McKinney (Jour. Washington Acad. Sci., *34*, 1944, 149), a genus originally differentiated from *Marmor* as containing viruses not yet inoculable save by tissue union; a natural group of viruses may be represented but their characteristics and affiliations seem not yet clear].

Genus II. **Acrogenus** *Holmes.*

(*Loc. cit.*, 110.)

Viruses of the Spindle-Tuber Group, inducing diseases characterized by abnormal growth habit of host plants without chlorotic or necrotic spotting, systemic chlorosis, witches'-broom formation, or production of galls. Generic name from Greek, meaning point- or peak-producing, in reference to shape of potatoes affected by potato spindle-tuber virus.

The type species is *Acrogenus solani* Holmes.

Key to the species of genus **Acrogenus.**

I. Infecting potato. 1. *Acrogenus solani.*

II. Infecting black currant. 2. *Acrogenus ribis.*

1. **Acrogenus solani** Holmes. (Handb. Phytopath. Viruses, 1939, 111.) From New Latin *Solanum*, generic name of potato.

Common names: Potato spindle-tuber virus, potato spindling-tuber virus, potato marginal leaf-roll virus.

Host: *SOLANACEAE—Solanum tuberosum* L., potato.

Geographical distribution: United States and Canada.

Induced disease: Plants erect, stiff, spindly, lacking vigor. Leaves small, erect, darker green than normal. Petioles sometimes slender, brittle. Tubers long, cylindrical, irregular in shape, tapered at ends, smooth and tender-skinned, of softer than normal flesh in spring. Eyes of tuber conspicuous.

Transmission: By inoculation of expressed juice; by use of contaminated knife in cutting successive tubers before planting; by contacts of freshly cut seed pieces. By aphids, *Myzus persicae* (Sulz.) and *Macrosiphum solanifolii* Ashm. (= *M. gei* Koch) (*APHIDIDAE*). Also by certain leaf-eating insects.

Thermal inactivation: At 60 to 65° C in 10 minutes (in tuber tissues).

Literature: Bald et al., Phytopath., *31*, 1941, 181–186; Folsom, Maine Agr. Exp. Sta., Orono, Bull. 312, 1923; Goss, Phytopath., *16*, 1926, 233, 299–303; *18*, 1928, 445–448; Nebraska Agr. Exp. Sta., Res. Bull. *47*, 1930; *53*, 1931; Jaczewski, La Défense des Plantes, Leningrad, *4*, 1927, 62–77 (Rev. Appl. Mycol., *6*, 1927, 572–573, Abst.); McLeod, Canad. Exp. Farms, Div. Bot., Rpt. for 1926, 1927.

Strains: A strain causing unmottled curly dwarf of potato has been given a varietal name to distinguish it from the type, var. *vulgaris* H. (*loc. cit.*, 111):

1a. *Acrogenus solani* var. *severus* H. (*loc. cit.*, 112). Inducing symptoms in potato on the whole more severe than those caused by the type strain.

Common name: Unmottled curly-dwarf strain of potato spindle-tuber virus. (Goss, Nebraska Agr. Exp. Sta., Res. Bull. 47, 1930; *53*, 1931; Schultz and Folsom, Jour. Agr. Res., *25*, 1923, 43–118.)

2. **Acrogenus ribis** H. (*loc. cit.*, 112). From Latin *ribes*, currant.

Common name: Black-currant reversion-disease virus.

Host: *SAXIFRAGACEAE—Ribes nigrum* L., European black currant.

Geographical distribution: British Isles.

Induced disease: In European black currant, leaves abnormally narrow and flat, small veins few. Flowers sometimes nearly transparent, smooth, sepals brightly colored beneath. Flowers and small fruits fall. Stems less woody than normal, with tendency to excessive gum production.

Transmission: By grafting. By big-bud mite, *Eriophyes ribis* (*ERIOPHYIDAE*). Not by inoculation of expressed juice. Not through soil. Not through seeds from diseased plants.

Literature: Amos and Hatton, Jour. Pom. and Hort. Science, *6*, 1926, 167–183; Amos et al., in East Malling Res. Sta., 15th Ann. Rpt., 1928, 43–46; Lee, Ann. Appl. Biol., *9*, 1935, 49–68.

Genus III. **Corium** *Holmes.*

(*Loc. cit.*, 119.)

Viruses of the Leaf-Roll Group, inducing diseases usually characterized by thicken-

ing and rolling of leaves. Foliage leathery. Sometimes conspicuous phloem necrosis. Generic name from Latin *corium*, leather.

The type species is *Corium solani* Holmes.

Key to the species of genus **Corium.**

I. Infecting potato. — 1. *Corium solani.*

II. Infecting beet. — 2. *Corium betae.*

III. Infecting raspberry. — 3. *Corium rubi.* 4. *Corium ruborum.*

1. **Corium solani** Holmes. (Handb. Phytopath. Viruses, 1939, 120.) From New Latin *Solanum*, generic name of potato.

Common name: Potato leaf-roll virus.

Hosts: *SOLANACEAE—Solanum tuberosum* L., potato. Experimentally, also other solanaceous species, *Datura stramonium* L., Jimson weed; *Lycopersicon esculentum* Mill., tomato; *Solanum dulcamara* L., bittersweet; *S. villosum.*

Insusceptible species: *CHENOPODIACEAE—Beta vulgaris* L., beet.

Geographical distribution: North America, France, British Isles; probably wherever potatoes are grown.

Induced disease: In potato, leaves thick, rigid, leathery, and rolled, their starch content excessive. Plants dwarfed. Tubers few, small, crisp. Tubers of some varieties show conspicuous phloem necrosis, germinate with spindling sprouts.

Transmission: By aphid, *Myzus persicae* (Sulz.) (*APHIDIDAE*), with incubation period of 24 to 48 hours. Also by *Myzus convolvuli* (Kalt.) (= *M. pseudosolani* Theob.), *M. circumflexus* (Buckt.), *Macrosiphum solanifolii* Ashm., and *Aphis abbreviata* Patch (*APHIDIDAE*). By grafting. Not by inoculation of expressed juice.

Literature: Artschwager, Jour. Agr. Res., *15*, 1918, 559–570; *24*, 1923, 237–245; Dykstra, *ibid.*, *47*, 1933, 17–32; Elze, Phytopath., *21*, 1931, 675–686; Folsom, Maine Agr. Exp. Sta., Bull. 297, 1921, 37–52; *410*, 1942, 215–250; Murphy, Scient. Proc. Roy. Dublin Soc., *17*, 1923, 163–184; Murphy and M'Kay, *ibid.*, *19*, 1929, 341–353; Schultz and Folsom, Jour. Agr. Res., *21*, 1921, 47–80; Smith, Ann. Appl. Biol., *16*, 1929, 209–229; *18*, 1931, 141–157; Stevenson et al., Am. Potato Jour., *20*, 1943, 1–10.

2. **Corium betae** *spec. nov.* From Latin *beta*, beet.

Common names: Sugar-beet yellows virus, beet-yellows virus, jaunisse virus, vergelingsziekte virus.

Hosts: *CHENOPODIACEAE—Beta vulgaris* L., beet; *B. maritima* L.; *B. cicla*; *Atriplex hortensis* L.; *A. sibirica* L.; *Chenopodium album* L., lamb's quarters; *Spinacia oleracea* L., spinach. *AMARANTHACEAE —Amaranthus retroflexus* L.

Insusceptible species: *SOLANACEAE—Solanum tuberosum* L., potato; all other tested solanaceous species.

Geographical distribution: Belgium, Netherlands, Denmark, England; perhaps Germany and the United States.

Induced disease: In beet, young leaves little affected; older leaves yellow, brittle, short, thick, containing excessive amounts of carbohydrates; necrosis in secondary phloem. In spinach, yellowing, necrosis between veins on old leaves.

Transmission: Not by inoculation of expressed juice. By aphids, *Myzus persicae* (Sulz.), *Aphis fabae* Scop., *Macrosiphum solanifolii* Ashm., and *Aulacor-*

thum solani Kalt. (*APHIDIDAE*); virus is not transmitted by these aphids to their descendants. Not through seeds of beet. Virus overwinters in beets stored for subsequent use in seed production.

Serological relationships: Specific precipitating antiserum effective with crude sap of diseased, not healthy, plants and with sap of diseased plants after passage through a Chamberland L_1, not L_3, filter candle; ineffective with sap from beet plants suffering from mosaic.

Thermal inactivation: Virus heated to about 52°C no longer precipitates with specific antiserum.

Literature: Kleczkowski and Watson, Ann. Appl. Biol., *31*, 1944, 116–120; Petherbridge and Stirrup, London, Ministry Agr. and Fisheries, Bull. *93*, 1935; Quanjer and Roland, Tijdschr. Plantenziekten, *42*, 1936, 45–70; Roland, *ibid.*, *45*, 1939, 1–22, 181–203; Schreven, Meded. Inst. voor Suikerbietenteelt, Bergen op Zoom, *6*, 1936, 1; Watson, Proc. Roy. Soc. London, Ser. B, *128*, 1940, 535–552; Ann. Appl. Biol., *29*, 1942, 358–365.

3. **Corium rubi** H. (*loc. cit.*, 121). From New Latin *Rubus*, generic name of raspberry, from Latin *rubus*, bramble bush.

Common name: Raspberry leaf-curl virus.

Host: *ROSACEAE—Rubus idaeus* L., red raspberry.

Insusceptible species: *ROSACEAE—Rubus occidentalis* L., black raspberry; *R. neglectus* Peck, purple raspberry.

Geographical distribution: United States, not in England.

Induced disease: In red raspberry, veins retarded in growth, causing downward curling of leaf margins and crinkling of leaf lamina. Foliage dark green, dry in appearance, not wilting readily. In late summer, leaves bronzed, leaf surface glistening. Diseased canes easily winterkilled. Berries small and poor. The English variety Lloyd George is intolerant of the disease and is killed.

Transmission: By aphid, *Aphis rubicola* Oestl. (= *A. rubiphila* Patch) (*APHIDIDAE*). Not by inoculation of expressed juice.

Literature: Bennett, Michigan Agr. Exp. Sta., Tech. Bull. 80, 1927; Phytopath., *20*, 1930, 787–802, Harris, East Malling Res. Sta., Ann. Rpt. for 1934, 1935; Rankin, New York Agr. Exp. Sta., Geneva, Tech. Bull. 175, 1931.

Strains: A strain differing from the type, var. *alpha* H. (*loc. cit.*, 121), has been given a varietal name derived from its common name, raspberry beta-curl virus:

3a. *Corium rubi* var. *beta* H. (*loc. cit.*, 122). Infecting black and purple raspberries, as well as the red raspberry, which alone is susceptible to the type strain, raspberry alpha-curl virus. (Bennett, Phytopath., *20*, 1930, 787–802.)

4. **Corium ruborum** (Zeller and Braun) *comb. nov.* (*Minuor ruborum* Zeller and Braun, Phytopath., *33*, 1943, 161.) From Latin *rubus*, bramble bush.

Common name: Raspberry decline-disease virus.

Host: *ROSACEAE—Rubus idaeus* L., red raspberry.

Geographical distribution: United States (Oregon).

Induced disease: In Cuthbert raspberry, shoots retarded in spring, reddish; leaves in autumn rolled downward, fluted along veins, less green than normal between veins, slightly bronzed along margins and crests between veins. Internodes shortened near tips of canes. Affected canes small, weak, not hardy in winter. Small roots and feeder rootlets fewer than in healthy plants. Disease progressive over about three years. Fruits small, irregular, tending to be globose, crumbly when ripe, worthless.

Transmission: By grafting. No insect vector is known.

Genus IV. Nanus *Holmes.*

(*Loc. cit.*, 123.)

Viruses of the Dwarf-Disease Group, inducing diseases characterized by dwarfing of host plants or by growth of adventitious shoots with short internodes; chlorotic mottling absent. Generic name from Latin *nanus*, dwarf.

The type species is *Nanus loganobacci* Holmes.

Key to the species of genus **Nanus.**

I. Infecting rosaceous plants.
  A. In loganberry and Phenomenal berry. 1. *Nanus loganobacci.*
  B. In black raspberry. 2. *Nanus orientalis.*
  C. In peach. 3. *Nanus mirabilis.*
  D. In ocean spray. 4. *Nanus holodisci.*
  E. In strawberry. 5. *Nanus fragariae.* 6. *Nanus cupuliformans.*
  F. In prune and plum. 7. *Nanus pruni.*

II. Infecting graminaceous plants.
  A. In sugar cane. 8. *Nanus sacchari.*

1. **Nanus loganobacci** Holmes. (Handb. Phytopath. Viruses, 1939, 124.)

From New Latin *loganobaccus*, specific epithet of loganberry, *Rubus loganobaccus* Bailey.

Common name: Loganberry-dwarf virus.

Hosts: *ROSACEAE—Rubus loganobaccus* Bailey, loganberry and Phenomenal berry.

Geographical distribution: United States (Oregon, Washington, and California).

Induced disease: In Phenomenal berry, leaves small, obovate, rigid, new canes short, spindly. In young plants, some necrosis along and between veins, leaves crinkled, finer veins chlorotic. Stems not streaked or mottled, normal in color. In late stages, canes very short, internodes short. Sepals and petals of flowers small. Fruit of fair size, but druplets ripen unevenly and tend to fall apart when picked. Loganberry is less susceptible than the Phenomenal berry but is similarly affected.

Transmission: By aphid, *Capitophorus tetrahodus* (*APHIDIDAE*). Not by inoculation of expressed juice.

Literature: Zeller, Phytopath., *15*, 1925, 732 (Abst.); *17*, 1927, 629–648.

2. **Nanus orientalis** H. (*loc. cit.*, 124). From Latin *orientalis*, eastern.

Common names: Raspberry-streak virus, raspberry eastern blue-stem virus, raspberry rosette virus.

Host: *ROSACEAE—Rubus occidentalis* L., black raspberry.

Insusceptible species: *ROSACEAE—Rubus idaeus* L., red raspberry; *R. phoenicolasius* Maxim., Japanese wineberry.

Geographical distribution: United States.

Induced disease: In black raspberry, plants stunted, becoming smaller in successive seasons; leaves usually curled, close together on canes, dark green, often twisted so as to be upside down. New canes show bluish violet dots, spots, or stripes near their bases and sometimes also on branches or on fruiting spurs.

Fruit inferior in size, quality, and quantity. Plants live only 2 or 3 years after infection on the average.

Strains: A strain of this virus is believed responsible for mild streak of black raspberries, in which purple to violet, greenish brown, or bluish streaks on canes are narrowly linear or elliptical in form and often very faint; when the bloom is rubbed off, the lesions appear as though watersoaked and discolored. Leaves are slightly curled, their veins cleared. Fruits are dry and dull in lustre, even while still red, and of poor flavor when ripe.

Literature: Bennett, Michigan Agr. Exp. Sta., Tech. Bull. 80, 1927; Wilcox U. S. Dept. Agr., Dept. Circ. 227, 1923; Woods and Haut, U. S. Dept. Agr., Plant Dis. Rpt., *24*, 1940, 338-340.

3. **Nanus mirabilis** H. (*loc. cit.*, 126). From Latin *mirabilis*, strange.

Common name: Peach phony-disease virus.

Hosts: *ROSACEAE—Prunus persica* (L.) Batsch, peach. Experimentally, also other *Prunus* species.

Geographical distribution: United States (Georgia, Alabama, Florida; sparsely also in Mississippi, Tennessee, South Carolina, Louisiana, Texas, Arkansas, Missouri).

Induced disease: In peach, tree dwarfed, foliage abnormally green, fruit small; flecks in wood, especially in roots; sections of roots show characteristic well-distributed purple spots after 3 to 5 minutes of treatment in 25 cc absolute methyl alcohol acidulated by the addition of 1 to 5 drops of concentrated, chemically pure hydrochloric acid.

Transmission: By root grafting, except by root-bark patch grafts, which are ineffective. Budding and grafting with parts of stem fail to transmit this virus.

Thermal inactivation: At 48° C in about 40 minutes in roots.

Literature: Hutchins, Georgia State Entomol. Bull., 78, 1933; Phytopath., *29*, 1939, 12 (Abst.); Hutchins and Rue, *ibid.*, *29*, 1939, 12 (Abst.).

4. **Nanus holodisci** H. (*loc. cit.*, 127). From New Latin *Holodiscus*, generic name of ocean spray.

Common name: Ocean-spray witches'-broom virus.

Host: *ROSACEAE—Holodiscus discolor* Max., ocean spray.

Geographical distribution: United States (Oregon and Washington).

Induced disease: In ocean-spray, diseased branches form clusters of thin wiry shoots with abnormally short internodes and crowded small leaves. Laterals numerous and more than normally branched. Bronzy red color acquired early by affected foliage.

Transmission: By aphid, *Aphis spiraeae* Schout. (*APHIDIDAE*). By grafting. Not demonstrated by inoculation of expressed juice.

Literature: Zeller, Phytopath., *21*, 1931, 923-925.

5. **Nanus fragariae** H. (Holmes, *loc. cit.*, 128; *Blastogenus fragariae* McKinney, Jour. Washington Acad. Science, *34*, 1944, 151.) From New Latin *Fragaria*, generic name of strawberry.

Common name: Strawberry witches'-broom virus.

Host: *ROSACEAE—Fragaria chiloensis* Duch. var. *ananassa* Bailey, cultivated strawberry.

Geographical distribution: United States (western Oregon).

Induced disease: In strawberry, leaves numerous, light in color, with spindly petioles, margins of leaflets bent down, runners shortened, plants dwarfed; flower stalks spindly and unfruitful; root systems normal and well developed.

Transmission: By aphis, *Myzus fragaefolii* Ckll. (*APHIDIDAE*). Not demonstrated by inoculation of expressed juice.

Literature: Zeller, Phytopath., *17*, 1927, 329-335.

6. **Nanus cupuliformans** Zeller and Weaver. (Phytopath., *31*, 1941, 851.) From diminutive of Latin *cupa*, tub, and participal from Latin *formare*, to form.

Common name: Strawberry-stunt virus.

Host: *ROSACEAE—Fragaria chiloensis* Duch. var. *ananassa* Bailey, cultivated strawberry.

Geographical distribution: United States (Oregon, Washington).

Induced disease: In strawberry, little if any reduction in chlorophyll, plants erect but short; leaves at first folded, later open, dull in lustre, with papery rattle when brushed by hand, leaflets cupped or with margins turned down, midveins tortuous; petioles ½ to ⅔ normal length; fruits small, usually hard and seedy; roots normal in appearance.

Transmission: By strawberry-leaf aphid, *Capitophorus fragaefolii* (*APHIDIDAE*). By grafting. Not by inoculation of expressed juice.

7. **Nanus pruni** H. (*loc. cit.*, 128). From New Latin *Prunus*, generic name of prune, from Latin *prunus*, plum tree.

Common name: Prune-dwarf virus.

Hosts: *ROSACEAE—Prunus domestica* L., prune and plum; var. *insititia* Bailey, the Damson plum, remains symptomless. Experimentally, also *Prunus persica* (L.) Batsch, peach.

Insusceptible species: *ROSACEAE—Prunus avium* L., cherry.

Geographical distribution: United States (New York); Canada (British Columbia, Ontario).

Induced disease: In prune, leaves small, narrow, rugose, distorted, glazed. Internodes short. Some branches escape and appear normal. Blossoms numerous, mature fruits few. Pistils aborted, petals narrow and distorted. In Damson and Bradshaw plums, no obvious manifestations of disease as a result of infection.

Transmission: By budding and other forms of grafting. Not demonstrated by inoculation of expressed juice. No insect vector is known.

Literature: Berkeley, Canada, Domin. Dept. Agr., Div. of Bot. and Plant Path., Science Service, Publ. 679, 1941; Hildebrand, Phytopath., *32*, 1942, 741–751; Thomas and Hildebrand, Phytopath., *26*, 1936, 1145–1148.

8. **Nanus sacchari** H. (*loc. cit.*, 129). From New Latin *Saccharum*, generic name of sugar cane, from Latin *saccharum*, sugar.

Common name: Sugar-cane sereh-disease virus.

Host: *GRAMINEAE—Saccharum officinarum* L., sugar cane.

Geographical distribution: Java, Borneo, Sumatra, Moluccas, India, Mauritius, Australia, Fiji, Formosa, Hawaii, Ceylon.

Induced disease: In sugar cane (Cheribon variety), plant dwarfed, shoots stunted, vascular bundles colored by the presence of a red gum; adventitious roots from many or all nodes.

Transmission: Not by inoculation of expressed juice. No insect vector is known.

Thermal inactivation: In cuttings of sugar cane, at 52° C in 30 minutes to 1 hour. Infected cane cuttings survive the heat treatment required for cure through inactivation of the causative virus.

Literature: Houtman, Arch. Suikerind. Nederland.-Indië, *33*, 1925, 631–642; Lyon, Bull. Exp. Sta. Hawaiian Sugar Planters' Assoc., Bot. Ser., *3*, 1921, 1–43; Wilbrink, Arch. Suikerind. Nederland.-Indië, *31*, 1923, 1–15.

Genus V. **Rimocortius** *Milbrath and Zeller.*

(Phytopath., *32*, 1942, 430.)

Viruses of the Rough-Bark Group, inducing diseases principally affecting bark, less often wood, leaves, or fruit. Generic name from Latin *rima*, cleft or fissure, and *cortex*, bark.

The type species is *Rimocortius kwanzani* Milbrath and Zeller.

(NOTE: The genus *Citrivir* (first named species, *Citrivir psorosis* Fawcett, Phytopath., *31*, 1941, 357) was proposed by its author as a *genus pro tempore* with the avowed purpose of accommodating viruses causing diseases in species of the plant-host genus *Citrus*. It appears to have been implied by the term *genus pro tempore* that evidences of natural relationship, when discovered, would permit even the first-named species of this genus to be assigned elsewhere. On the assumption that a permanent genus is nothing more than a *type species* and such other species as may be added to it by one or another author, it must be felt that a *genus pro tempore*, however convenient as an expedient, cannot become a permanent genus under any circumstances, because its first-named species would appear not to be a permanent part of the genus and so intended not to be a true type-species. Without a type species there would seem to be no permanent genus concept.

The system by which the term *Citrivir* was coined (explained by its author as use of the genitive of the host-genus name, *Citris*, plus *vir*, signifying virus) seems in itself acceptable, for it is commonly agreed that a generic name may be made in an arbitrary manner. It may be noted that use of the stem of the host-genus name (*Citr-*) with connecting vowel *i* and suffix *-vir*, possibly a more orthodox procedure, would have given the same result in the present instance. The original definition of the term *Citrivir* might be thought to be repugnant as disregarding concepts of natural interspecific relationships that are essential to the spirit of binomial nomenclature. Were the genus to be regarded as permanent rather than *pro tempore*, however, the scope of the genus would come to be wholly changed by usage, when, with passage of time related species would be added to what in this case would be a type species, without regard to the unorthodox intent of the original definition but solely in accordance with similarities between viruses. A generic concept need never be accepted as rigidly defined, whether initially, as has been attempted in this case, or upon further experience, because a genus may still grow by the addition of closely allied new species beyond any limit that may be set. On this account an original, or any subsequent, definition may be regarded as subject to unlimited change so long as the type species is logically retained. The form and definition of the term *Citrivir* would not, therefore, militate against its continued use. Its avowedly temporary status alone seems decisively to do so.

The originally monotypic genus *Rimocortius*, published in the following year, was defined only by the combined generic and specific description, and was not referred to a family by its authors. The type, because at first the only species, *Rimocortius kwanzani*, is the flowering-cherry rough-bark virus. This type species might well be associated with the species *Citrivir psorosis*, citrus-psorosis virus, discussed above, both affecting bark principally, though foliage also to some extent. Although the genus *Citrivir* was named in 1941 and *Rimocortius* not until 1942, the first was intended as a temporary assemblage only, as above indicated. It would seem appropriate, therefore, to include the virus that was known temporarily as *Citrivir psorosis* in the permanent genus *Rimocortius* Milbrath and Zeller and to assign this genus to the family *Marmoraceae*.)

Key to the species of the genus **Rimocortius.**

I. Affecting cherry.
1. *Rimocortius kwanzani.*

II. Affecting *Citrus*.
2. *Rimocortius psorosis.*

III. Affecting pear.
3. *Rimocortius pyri.*

1. **Rimocortius kwanzani** Milbrath and Zeller. (Phytopath., *32*, 1942, 430.) From Kwanzan, name of a variety of flowering cherry.

Common name: Flowering-cherry rough-bark virus.

Hosts: *Prunus serrulata* Lindl. var. Kwanzan, flowering cherry; *P. avium* L., Mazzard cherry.

Geographical distribution: United States (Oregon).

Induced disease: In flowering cherry, tree dwarfed, deficient in lateral branches; bark deep brown, roughened, splitting longitudinally; internodes shortened, bunching leaves; leaves arched downward; midribs of leaves split and cracked on under surface. In Mazzard cherry, no manifestation of disease, but carrier condition; budded Mazzard stock may transmit disease to healthy Kwanzan cherry cions.

Transmission: By budding, generally even if the inserted bud fails to survive.

Literature: Milbrath and Zeller, Phytopath., *32*, 1942, 428–430; Thomas, *ibid.*, *32*, 1942, 435–436.

2. **Rimocortius psorosis** (Fawcett) *comb. nov.* (*Citrivir psorosis* Fawcett, Phytopath., *31*, 1941, 357.) Specific name meaning "of the disease known as psorosis."

Common name: Citrus-psorosis virus.

Hosts: *RUTACEAE—Citrus sinensis* Osbeck, orange; *C. limonia* Osbeck, lemon; *C. maxima* Merr., grapefruit.

Geographical distribution: World-wide where citrus trees are grown.

Induced disease: In citrus, small, elongated, light colored areas or flecks in the region of small veins on young, tender foliage; leaves sometimes warped; (chlorotic?) clearing of veins, and chlorotic line patterns, sometimes concentric. Outer layers of bark scale away; depressions and deformities appear in bark and wood. Lemons, as a rule, are more tolerant than oranges and are not subject to the bark changes.

Transmission: By grafting, including root grafting and patch bark grafting. Not by inoculation of expressed juice. No insect vector is known.

Literature: Bitancourt et al., Phytopath., *33*, 1943, 865–883; Fawcett, *ibid.*, *24*, 1934, 659–668; Science, *92*, 1940, 559–561; Phytopath., *31*, 1941, 356–357; Fawcett and Bitancourt, *ibid.*, *33*, 1943, 837–864; Rhoads, *ibid.*, *32*, 1942, 410–413; Webber and Fawcett, Hilgardia, *9*, 1935, 71–109.

Strains: Three strains differing from the type have been recognized. The type, var. *vulgare* Fawcett, Phytopath., *31*, 1941, 357, causes psorosis A, the common scaly-bark type of disease, with pustular eruptions of outer layers of bark in limited areas, with or without exudation of gum; later a drab-gray, cinnamon-drab to rufus discoloration of the wood, accompanied by decline of the affected tree. Others, that contrast with the type, are:

2a. *Rimocortius psorosis* var. *anulatum* Fawcett. (Phytopath., *31*, 1941, 357.) From Latin *anulatus*, with a ring. Causing psorosis B, known from California, resembling zonate chlorosis of Brazil in effects on leaves and fruits. Psorosis B is characterized by rapid scaling of outer bark in continuous areas, progressing rapidly along one side of trunk or branch; gum exudes in advance of scaling, necrosis follows; large circular discolored and corky spots, sometimes concentric, on fruits and mature leaves; on some fruits, circular or semi-circular furrows and bumps; rapid decline of the affected tree.

2b. *Rimocortius psorosis* var. *concavum* Fawcett and Bitancourt. (Phytopath., *33*, 1943, 850.) From Latin *concavus*, concave. Causing concave-gum psorosis, characterized by concavities of various sizes on trunks and larger limbs of affected trees, often by zonate patterns on young leaves during periods of rapid growth.

2c. *Rimocortius psorosis* var. *alveatum* Fawcett and Bitancourt. (Phytopath., *33*, 1943, 854.) From Latin *alveatus*, hollowed out like a trough. Causing blind-pocket psorosis, characterized by trough-like pockets in bark and wood or by eruptive lesions.

3. **Rimocortius pyri** (Holmes) *comb. nov.* (*Marmor pyri* Holmes, Handb. Phytopath. Viruses, 1939, 76.) From New Latin *Pyrus*, generic name of pear, from Latin *pirus*, pear tree.

Common name: Pear stony-pit virus.

Host: *ROSACEAE—Pyrus communis* L., pear.

Geographical distribution: United States (Oregon, Washington, California).

Induced disease: In pear, fruit deeply pitted and deformed; bark cracked and resembling oak bark; veinlet chlorosis of some leaves, failure of lateral buds to grow, reduction of foliage. Bartlett and Comice varieties of pear appear to be tolerant, producing sound fruit from infected trees.

Transmission: By budding. Not by inoculation of expressed juice. No insect vector is known.

Literature: Kienholz, Phytopath., *29*, 1939, 260–267; *30*, 1940, 787 (Abst.).

Genus VI. Adelonosus *Brierley and Smith.*

(Phytopath., *34*, 1944, 551.)

Viruses capable of multiplying in living plants but producing no recognizable symp toms in these except on interaction with distinct viruses with which they form complexes. Transmitted by aphids, by sap, or by both means. Generic name from Greek *adelos*, invisible, and *nosos*, disease. Only one species is recognized thus far; this is the type species, *Adelonosus lilii* Brierley and Smith.

1. **Adelonosus lilii** Brierley and Smith. (Phytopath., *34*, 1944, 551.) From Latin *lilium*, lily.

Common name: Lily-symptomless virus.

Host: *LILIACEAE—Lilium longiflorum* Thunb., Easter lily.

Insusceptible species: All other tested lilies and many related plants in the same and other families (for list, see Phytopath., *34*, 1944, 549).

Geographical distribution: United States, Japan; probably coextensive with commercial culture of Easter lily.

Induced disease: In Easter lily, no obvious manifestation of disease when this virus is present alone; when together with cucumber-mosaic virus, however, the lily-symptomless virus is a determining factor in the production of necrotic-fleck disease; the lily-symptomless virus is so widely distributed in supposedly healthy stocks of the Easter lily that cucumber-mosaic virus formerly was thought to be the sole determining factor in necrotic flecking, now recognized to be caused by the virus complex lily-symptomless virus (*Adelonosus lilii*) *plus* cucumber-mosaic virus (*Marmor cucumeris*); the complex acts independently of the presence or absence of lily latent-mosaic virus (*Marmor mite*), which is often present with the essential members of the complex in flecked Easter lilies.

Transmission: By inoculation of expressed juice, with some difficulty. By aphid, *Aphis gossypii* Glov., cotton aphid (*APHIDIDAE*); preinfective period after obtaining virus, 4 to 6 days.

FAMILY III. ANNULACEAE HOLMES.

(Handb. Phytopath. Viruses, 1939, 97.)

Viruses of the Ringspot Group, causing diseases usually characterized by necrotic or chlorotic spotting with concentric-ring lesions and eventual recovery from obvious disease with non-sterile immunity. Hosts, higher plants; vectors unknown. There is a single genus.

Genus I. Annulus *Holmes.*

(*Loc. cit.*, 97.)

Characters those of the family. Generic name from Latin *annulus*, a ring. The type species is *Annulus tabaci* Holmes.

Key to the species of genus **Annulus.**

I. Found occurring naturally in the Western Hemisphere.

A. In tobacco.

1. *Annulus tabaci.*
2. *Annulus zonatus.*
3. *Annulus orae.*
4. *Annulus apertus.*

B. In potato.

5. *Annulus dubius.*

C. In delphinium.

6. *Annulus delphinii.*

II. Old World species.

7. *Annulus bergerae.*

1. **Annulus tabaci** Holmes. (Handb. Phytopath. Viruses, 1939, 98; *Marmor anularium* McKinney, Jour. Washington Acad. Sci., *34*, 1944, 327.) From New Latin *Tabacum*, early generic name for tobacco.

Common names: Tobacco-ringspot virus, green ringspot virus, yellow ringspot virus, ring spot No. 1 virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., *Petunia violacea* Lindl., *Solanum tuberosum* L. *CUCURBITACEAE—Cucumis sativus* L. Experimentally this virus has been found capable of infecting many species of plants in a large number of families; these include all tested species of the *SOLANACEAE*, *SCROPHULARIACEAE*, *COMPOSITAE*, and *CUCURBITACEAE*. Many species of the *LEGUMINOSAE* are susceptible and one, *Vigna sinensis* (L.) Endl., is used as an indicator plant for quantitative measurement because it displays conspicuous reddish-brown necrotic lesions around points of initial infection.

Geographical distribution: United States.

Induced disease: In tobacco, necrotic ring-like primary lesions, followed by secondary necrotic rings on younger leaves. Subsequently, affected plants recover. After recovery from obvious disease, virus content of plants is only 10 to 20 per cent of that of recently infected plants. Some varieties may show mosaic-like patterns in young leaves at 16°C.

Transmission: By inoculation of expressed juices. Through about 20 per cent of seeds from diseased petunia plants. Not by dodder, *Cuscuta campestris* Yuncker (*CONVOLVULACEAE*).

Serological relationships: Induces the formation of specific precipitating antibodies when injected into bloodstream of rabbit.

Immunological relationships: Recovered tobacco plants are not susceptible to reinfection with this virus but are readily infected with *Annulus zonatus* or *A. orae*. This virus produces primary lesions on

leaves of plants immune to reinfection with *A. bergerac.*

Thermal inactivation: At 68° C in 10 minutes.

Filterability: Passes V, N, and perhaps W Berkefeld filters.

Other properties: Particle size estimated by filtration experiments as about 19 millimicrons. Sedimentation constant, $S_{20}^{\circ} = 115 \times 10^{-13}$ cm. sec.$^{-1}$ dyne^{-1}. Infective in dilutions of 10^{-7} after purification. Inactivated in 1 hour below pH 3 or above pH 10.8. Recovered plants of tobacco contain 0.002 mg of virus per gram, recently infected plants about 6 times as much. Optimum conditions for retaining infectivity of stored virus include suspension in 0.01 M phosphate buffer at pH 7 and storage at 4° C.

Literature: Fenne, Phytopath., *21*, 1931, 891–899; Fromme et al., *ibid.*, *17*, 1927, 321–328; Henderson, *ibid.*, *21*, 1931, 225–229; Henderson and Wingard, Jour. Agr. Res., *43*, 1931, 191–207; Price, Contrib. Boyce Thompson Inst., *4*, 1932, 359–403; Phytopath., *26*, 1936, 503–529; Am. Jour. Bot., *27*, 1940, 530–541; Am. Naturalist, *74*, 1940, 117–128; Priode, Am. Jour. Bot., *15*, 1928, 88–93; Stanley, Jour. Biol. Chem., *129*, 1939, 405–428, 429–436; Stanley and Wyckoff, Science, *85*, 1937, 181–183; Valleau, Kentucky Agr. Exp. Sta., Bull. 327, 1932; Wingard, Jour. Agr. Res., *37*, 1928, 127–153; Woods, Contrib. Boyce Thompson Inst., *5*, 1933, 419–434.

Strains: A number of distinctive strains have been collected in nature and studied experimentally. The following have been given varietal names to distinguish them from the type, var. *virginiensis* H., *loc. cit.*, 98:

1a. *Annulus tabaci* var. *kentuckiensis* H. (*loc. cit.*, 99). Differing from the typical strain in producing less necrosis and less stunting in tobacco. (Price, Phytopath., *26*, 1936, 665–675; Valleau, Kentucky Agr. Exp. Sta., Bull. 327, 1932.)

1b. *Annulus tabaci* var. *auratus* H. (*loc. cit.*, 100). Secondary lesions in tobacco at first yellow spots or rings, becoming necrotic subsequently. Recovery less complete than with type, abnormal yellowing of old leaves tending to persist. (Chester, Phytopath., *25*, 1935, 686–701; Price, Phytopath., *26*, 1936, 665–675; Valleau, Kentucky Agr. Exp. Sta., Bull. 327, 1932; Phytopath., *29*, 1939, 549–551.)

2. **Annulus zonatus** H. (*loc. cit.*, 101). From Latin *zonatus*, zonate.

Common names: Tomato-ringspot virus, ring spot No. 2 virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., tobacco. Experimentally this virus has been found to infect many species of plants in a large number of families.

Geographical distribution: United States.

Induced disease: In tobacco, zonate necrotic primary lesions and, temporarily, secondary lesions of the same type; recovery with specific, non-sterile immunity. In tomato, systemic infection, yellowish-green or necrotic ring-like lesions; stunting.

Transmission: By inoculation of expressed juice.

Immunological relationships: Recovered plants are immune to reinfection but are still susceptible to *Annulus tabaci*, *A. bergerac*, and several mosaic-type viruses that have been tested.

Thermal inactivation: At 55 to 60° C in 10 minutes.

Filterability: Passes Gradocol membrane 100 millimicrons in average pore diameter. Particle size estimated as 50 millimicrons or less.

Literature: Price, Phytopath., *26*, 1936 665–675; Am. Jour. Bot., *27*, 1940, 530–541.

3. **Annulus orae** H. (Holmes, *loc. cit.*, 103; *Tractus orae* Valleau, Phytopath., *30*, 1940, 826.) From Latin *ora*, edge, in reference to occurrence of induced disease near edge of tobacco fields.

Common name: Tobacco-streak virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., tobacco. Experimentally, a

number of other solanaceous plants have been reported as susceptible, but not *Capsicum frutescens* L., pepper; *Lycopersicon esculentum* Mill., tomato; *Solanum melongena* L., eggplant; or *S. tuberosum* L., potato.

Geographical distribution: United States.

Induced disease: In tobacco, local and systemic necrosis in 3 days, with irregular spot, line, and ring-like lesions, followed by recovery from necrotic manifestations of disease. Recovered leaves may show a mild mottling and regularly contain virus; reinoculation does not induce formation of necrotic lesions in them.

Transmission: By inoculation of expressed juice. Not through seeds from diseased plants.

Immunological relationships: No cross-protection with respect to *A. tabaci*, and several viruses of the mosaic group.

Thermal inactivation: At 53° C in 10 minutes.

Literature: Johnson, Phytopath., *26*, 1936, 285–292; Trans. Wisconsin Acad. Sciences, Arts and Letters, *30*, 1937, 27–34.

4. **Annulus apertus** *spec. nov.* From Latin *apertus*, frank.

Common name: Broad-ringspot virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., tobacco. Experimentally also to many species in this and other families.

Insusceptible species: *CHENOPODIACEAE—Beta vulgaris* L. *CUCURBITACEAE—Citrullus vulgaris* Schrad. *LEGUMINOSAE—Medicago sativa* L., *Melilotus alba* Desr.

Geographical distribution: United States (Wisconsin).

Induced disease: In tobacco, indistinct yellow-spot primary lesions, becoming chlorotic or necrotic rings with concentric markings; small chlorotic rings, sometimes concentric, or fine brown necrotic rings as secondary lesions; young leaves puckered at first, somewhat malformed.

Transmission: By inoculation of expressed juice.

Immunological relationships: Protects against reinfection with homologous virus but leaves host susceptible to infection by *Annulus tabaci*, *A. zonatus*, and some mosaic-type viruses.

Literature: Johnson and Fulton, Phytopath., *32*, 1942, 605–612.

5. **Annulus dubius** (Holmes) *comb.nov.* (*Marmor dubium* H., *loc. cit.*, 42.) From Latin *dubius*, uncertain, in reference to a common name, potato virus X, often used to designate this virus.

Common name: Potato-mottle virus (strains of this virus have been studied at various times under the names potato latent virus, potato virus X, potato-anecrosis virus, virulent latent virus, simple mosaic virus, healthy potato virus, Hyoscyamus IV virus, President streak virus, potato foliar-necrosis virus, potato acronecrotic streak virus, Up-to-Date streak virus, potato viruses B and D, Solanum viruses 1, 4, and 6.)

Hosts: *SOLANACEAE—Solanum tuberosum* L., potato; *Lycopersicon esculentum* Mill., tomato. Experimentally, also *SOLANACEAE—Capsicum frutescens* L., pepper; *Datura stramonium* L., Jimson weed; *Hyoscyamus niger* L., henbane; *Nicotiana tabacum* L., tobacco; *Physalis alkekengi* L.; *Solanum dulcamara* L., bittersweet; *S. nigrum* L., black nightshade. *AMARANTHACEAE—Amaranthus retroflexus* L. *COMPOSITAE—Chrysanthemum morifolium* Ram. *SCROPHULARIACEAE—Veronica* sp., common speedwell.

Geographical distribution: Widespread throughout the world; present in all known stocks of tubers of some potato varieties in the United States.

Induced disease: In potato, usually no chlorotic mottling, sometimes a little; intracellular inclusions of the vacuolated, granular type; some varieties that are virtually immune in the field owe their tendency to localize the virus in necrotic primary lesions or in top-necrosis of first systemically infected plants to a dom-

inant allele of a gene *nx*, which characterizes plants showing a mosaic of some degree of intensity on infection with this virus; the variety known as S41956 is immune to all tested strains of the virus and possesses two dominant genes both required for resistance. In tomato, systemic mild chlorotic mottling; if a strain of tobacco-mosaic virus is also present, a severe systemic necrosis, known as double-virus streak, is induced.

Transmission: By inoculation of expressed juice. Experimentally, by leaf contacts mainly under the influence of wind. No insect vector is known. Not transmitted through true seeds of the potato.

Serological relationships: Cross precipitin reactions between constituent strains of this virus. No cross reaction with potato aucuba-mosaic, potato mild-mosaic, potato-veinbanding, tobacco-mosaic, tobacco-etch, tobacco-ringspot or pea-mosaic virus. Antisera prepared by injecting rabbits intravenously with virus inactivated by nitrous acid, like those prepared with active virus, fix complement and flocculate with virus suspensions (though not with juice of healthy host plants); they are also effective in neutralizing the virus.

Immunological relationships: Tobacco and *Datura* plants infected by the type strain of this virus become immune to the more severe potato-ringspot strain. No protection against the severe strain is afforded by previous infection with tobacco-mosaic, tobacco-ringspot, tomato spotted-wilt, or cucumber-mosaic virus.

Thermal inactivation: At 70° C. in 10 minutes.

Filterability: Passes Pasteur-Chamberland L_1, L_2, and L_4 filters.

Other properties: Digested by 0.02 per cent solution of pepsin in 3 hours at pH 4, at 38° C. Digested also by trypsin. Inactivated by papaine and cyanide, but by neither separately. Isoelectric point near pH 4. Dilute solutions show anisotropy of flow. Concentrated solutions are spontaneously birefringent. Properties of the type strain have been less studied than those of the potato-ringspot strain of this virus.

Literature: Bawden, Proc. Roy. Soc. London, Ser. B, *116*, 1934, 375–395; Bawden and Pirie, Brit. Jour. Exp. Path., *17*, 1936, 64–74; Bawden et al., *ibid.*, *17*, 1936, 204–207; Blodgett, Phytopath., *17*, 1927, 775–782; Böhme, Phytopath. Ztschr., *6*, 1933, 517–524; Cadman, Jour. Genetics, *44*, 1942, 33–52; Chester, Phytopath., *27*, 1937, 903–912; Clinch, Sci. Proc. Roy. Dublin Soc., *23*, 1942, 18–34; Johnson, Wisconsin Agr. Exp. Sta., Res. Bull. 63, 1925; Koch, Phytopath., *23*, 1933, 319–342; Köhler, Phytopath. Ztschr., *5*, 1933, 567–591; *7*, 1934, 1–30; Loughnane and Murphy, Nature, *141*, 1938, 120; van der Meer, Cent. f. Bakt., II Abt., *87*, 1932, 240–262; Salaman, Nature, *131*, 1933, 468; Schultz et al., Phytopath., *27*, 1937, 190–197; *30*, 1940, 944-951; Spooner and Bawden, Brit. Jour Exp. Path., *16*, 1935, 218–230; Stevenson et al., Phytopath., *29*, 1939, 362-365.

Strains: Several variants of potato-mottle virus, differing from the type, var. *vulgaris* H. (*loc. cit.*, 42), have been recognized as distinctive varieties under the following names:

5a. ***Annulus dubius*** var. *annulus* H. (*loc. cit.*, 44). From Latin *annulus*, ring.

Common name: Ringspot strain of potato-mottle virus. Necrotic primary and secondary ring-like lesions in experimentally infected tobacco plants. Indistinguishable from type strain by ordinary precipitin test, but distinguishable when appropriately absorbed sera are used. This strain has been more frequently studied than the type. Juice of infected tobacco plants contains about 0.02 to 0.10 mg of virus per ml. Sedimentation constants, $S_{20^\circ} = 113 \times 10^{-13}$ and 131×10^{-13} cm. sec.$^{-1}$ dyne^{-1}. Dissymmetry constant 2.78. Molecular weight 26×10^6. Particle size estimated to be 433 by 9.8 millimicrons, 43.9 times as long as wide. Isoelectric point near pH 4. Stable between pH 4 and pH 9.5. Concentrated solutions are spontaneously birefringent. Dilute solutions show

anisotropy of flow. Destroyed by drying. Inactivated by papaine and cyanide, but by neither separately. Digested by 0.02 per cent solution of pepsin in 3 hours at pH 4, at 38° C. Digested also by trypsin. About 6 per cent of the purified virus is reported to be a pentose nucleic acid, but the carbohydrate to phosphorus ratio is about twice that for yeast nucleic acid. Guanine and pentose present. Analysis of sedimented virus, carbon 47.7 to 49.5 per cent, hydrogen 6.8 to 7.7 per cent, nitrogen 14.6 to 17.0 per cent, phosphorus 0.4 to 0.7 per cent, sulfur 1.1 per cent, carbohydrate 2.5 to 4.3 per cent, ash 2.0 to 2.5 per cent. Reduction of carbohydrate content of sample to 2.5 per cent does not reduce activity of virus; further reduction inactivates. (Ainsworth, Ann. Appl. Biol., *21*, 1934, 581–587; Bawden, Brit. Jour. Exp. Path., *16*, 1935, 435–443; Bawden and Pirie, *ibid.*, *19*, 1938, 66–82; Birkeland, Bot. Gaz., *95*, 1934, 419–436; Chester, Phytopath., *26*, 1936, 778–785; Johnson, Wisconsin Agr. Exp. Sta., Res. Bull. *76*, 1927; Loring, Jour. Biol. Chem., *126*, 1938, 455–478; Loring and Wyckoff, *ibid.*, *121*, 1937, 225–230.)

5b. *Annulus dubius* var. *flavus* H. (*loc. cit.*, 46). From Latin *flavus*, yellow.

Common name: Yellow-mottle strain of potato-mottle virus. Differing from the type by imparting a yellow cast to foliage of infected potatoes. (Putnam, Canad. Jour. Res., Sec. C, *15*, 1937, 87–107.)

5c. *Annulus dubius* var. *obscurus* H. (*loc. cit.*, 46). From Latin *obscurus*, obscure. Common name: Masked-mottle strain of potato-mottle virus. Differing from the type by systemically infecting potato, tobacco, and Jimson weed without symptoms under ordinary experimental conditions; in pepper, however, systemic necrosis is induced, as by all known strains. (Chester, Phytopath., *26*, 1936, 778–785.)

6. **Annulus delphinii** *spec. nov.* From New Latin *Delphinium*, generic name of host.

Common names: Delphinium-ringspot virus, perennial-delphinium ringspot virus.

Hosts: *RANUNCULACEAE—Delphinium* sp., perennial delphiniums. Experimentally, also to *CHENOPODIACEAE—Beta vulgaris* L. *CUCURBITACEAE—Cucumis sativus* L., cucumber. *MALVACEAE — Gossypium hirsutum* L. *RANUNCULACEAE—Ranunculus asiaticus* L. (symptomless carrier). *SOLANACEAE—Datura stramonium* L., *Nicotiana alata* Link and Otto, *N. glutinosa* L., *N. rustica* L., *N. tabacum* L., *Petunia hybrida* Vilm.

Geographical distribution: United States (California).

Induced disease: In perennial delphiniums, faint chlorotic rings with green or yellow centers on young leaves; irregular chlorotic spots, yellow bands, or irregular chlorotic rings on mature leaves.

Transmission: By inoculation of expressed juice in the presence of finely powdered carborundum.

Thermal inactivation: At 65° C in 10 minutes.

Literature: Severin and Dickson, Hilgardia, *14*, 1942, 465–490.

7. **Annulus bergerac** H. (*loc. cit.*, 102). From Bergerac, a town in southwest France.

Common name: Bergerac-ringspot virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., tobacco. Experimentally, this virus has been transferred to several other solanaceous plants and to *Phaseolus vulgaris* L., bean, in the family *LEGUMINOSAE*.

Geographical distribution: France.

Induced disease: In tobacco, thin necrotic-ring primary lesions, followed by

systemic mottling with some chlorotic rings on the dark green islands. Later, complete recovery occurs, with non-sterile immunity.

Transmission: By inoculation of expressed juice.

Immunological relationships: Recovered plants are susceptible to infection by *Annulus tabaci* and *A. zonatus*.

Thermal inactivation: At 80° C in 10 minutes.

Literature: Smith, A textbook of plant virus diseases, P. Blakiston's Son and Co., Inc., Philadelphia, 1937, 285–289.

FAMILY IV. RUGACEAE HOLMES.

(Handb. Phytopath. Viruses, 1939, 114)

Viruses of the Leaf-Curl Group, causing diseases characterized by suddenly arrested development of invaded tissues, resulting in leaf curl, enations, and other deformities. Vectors, typically white-flies (*ALEYRODIDAE*). There is a single genus.

Genus I. Ruga *Holmes.*

(*Loc. cit.*, 114.)

Characters those of the family. Generic name from Latin *ruga*, a wrinkle. The type species is *Ruga tabaci* Holmes.

Key to the species of genus **Ruga.**

I. Infecting tobacco. — 1. *Ruga tabaci.*

II. Infecting cotton. — 2. *Ruga gossypii.*

III. Infecting cassava (*Manihot*). — 3. *Ruga bemisiae.*

IV. Infecting sugar-beet. — 4. *Ruga verrucosans.*

1. **Ruga tabaci** Holmes. (Handb. Phytopath. Viruses, 1939, 115.) From New Latin *Tabacum*, former generic name of tobacco.

Common names: Tobacco leaf-curl virus, kroepoek virus, curl-disease virus, crinkle-disease virus.

Hosts: *SOLANACEAE—Nicotiana tabacum* L., tobacco. *COMPOSITAE—Vernonia iodocalyx, V. cineria, Ageratum conyzoides* L., *Synedrella nodiflora* Gaertn. Experimentally, also other solanaceous plants.

Insusceptible species: *MALVACEAE—Gossypium hirsutum* L., cotton.

Geographical distribution: Tanganyika, Southern Rhodesia, Southern Nigeria, Nyasaland, India, Sumatra, Formosa.

Induced disease: In tobacco, leaves curled and crinkled, with occasional leafy outgrowths or enations. Veins greened and thickened. No chlorosis nor necrosis. Plant stunted.

Transmission: By white-fly, *Bemisia gossypiperda* Misra and Lamba (*ALEYRODIDAE*). By grafting. Not by inoculation of expressed juice.

Literature: Kerling, Phytopath., *23*, 1933, 175-190; Mathur, Indian Jour. Agr. Sci., *3*, 1933, 89-96; Matsumoto and Tateoko, Trans. Nat. Hist. Soc. Formosa, *30*, 1940, 31-33; Pal and Tandon, Indian Jour. Agr. Sci., *7*, 1937, 363-393; Pruthi and Samuel, *ibid.*, *7*, 1937, 659-670; Storey, Nature, *128*, 1931, 187-188; East African Agr. Jour., *1*, 1935, 148-153; Thung, Meded. Proefsta. Vorstenl. Tabak Java, *72*, 1932; *78*, 1934.

2. **Ruga gossypii** H. (*loc. cit.*, 116). From Latin *gossypium*, cotton.

Common names: Cotton leaf-curl virus, cotton leaf-crinkle virus.

Hosts: *MALVACEAE—Gossypium hirsutum* L., cotton; *G. peruvianum* Cav.; *G. vitifolium* Lam.; *Hibiscus cannabinus* L.; *H. esculentus* L.; *H. sabdariffa* L.; *Althaea rosea* Cav., hollyhock; Sakel (hybrid) cotton.

Geographical distribution: The Sudan and Nigeria, in Africa.

Induced disease: In cotton, clearing of veins, blistering and pale spotting of leaves; leaves puckered at edge and unsymmetrical. Internodes shortened, producing bunchy growth.

Transmission: By white-fly, *Bemisia gossypiperda* Misra and Lamba (*ALEY-*

RODIDAE). Not through egg of insect vector. Not by inoculation of expressed juice. Not through soil. Not through seeds from diseased plants.

Literature: Bailey, Empire Cotton Growing Rev., *11*, 1934, 280; Kirkpatrick, Bull. Entom. Res., *21*, 1930, 127–137; *22*, 1931, 323–363.

3. **Ruga bemisiae** H. (Holmes, *loc. cit.*, 117; *Ochrosticta bemisiae* McKinney, Jour. Washington Acad. Sci., *34*, 1944, 149.) From New Latin *Bemisia*, generic name of insect vector.

Common names: Cassava-mosaic virus, cassava Kräuselkrankheit virus.

Hosts: *EUPHORBIACEAE—Manihot utilissima* Pohl, cassava; *M. palmata* Muell.; *M. dulcis*.

Geographical distribution: Gold Coast, Belgian Congo, French Cameroons, Rhodesia, Liberia, Madagascar, probably throughout Africa and adjacent islands; Java.

Induced disease: In *Manihot utilissima*, leaves unsymmetrical, curled, distorted, mottled; internodes shortened, plants stunted. Axillary buds produce an extra number of side branches.

Transmission: By white-flies (*ALEYRODIDAE*), *Bemisia nigeriensis* Corb., in Southern Nigeria, and *B. gossypiperda* Misra and Lamba, in Belgian Congo and Tanganyika. White-flies infect only young leaves. Not by needle-puncture, rubbing, or hypodermic-needle injection of juice expressed from diseased plants.

Literature: Dade, Yearbk. Dept. Agr. Gold Coast, 1930, 245; Dufrénoy and Hedin, Rev. Bot. Appl., *9*, 1929, 361–365; Golding, Trop. Agric., Trinidad, *13*, 1936, 182–186; Kufferath and Ghesquière, Compt. rend. Soc. Biol. Belge, *109*, 1932, 1146; Lefevre, Bull. Agr. Congo Belge, *26*, 1935, 442; McKinney, Jour. Agr. Res., *39*, 1929, 557–578; Muller, Bull. Inst. Plantenziekt., *24*, 1931, 1–17; Pascalet, Agron. Colon., *21*, 1932, 117; Staner, Bull. Agr. Congo Belge, *22*, 1931, 75; Storey, East Afr. Jour., *2*, 1936, 34–39; Storey and Nichols, Ann. Appl. Biol., *25*, 1938, 790–806; Zimmermann, Pflanzer, *2*, 1906, 145.

4. **Ruga verrucosans** Carsner and Bennett. (*Chlorogenus eutetticola* (in error for *eutettigicola*, from New Latin *Eutettix*, genus name of a vector, and Latin *-cola*, dweller in or inhabitant of) Holmes, 1939, *loc. cit.*, 11; Carsner and Bennett, Science *98*, 1943, 386.) From Latin, meaning: causing rough swellings.

Common name: Sugar-beet curly-top virus.

Hosts: Very wide range in many families of dicotyledonous plants. Among the horticulturally important host plants are the sugar beet (*Beta vulgaris* L., *CHENOPODIACEAE*); bean (*Phaseolus vulgaris* L., *LEGUMINOSAE*); squash (*Cucurbita* species, *CUCURBITACEAE*); and tomato (*Lycopersicon esculentum* Mill., *SOLANACEAE*).

Geographical distribution: Western North America; in Argentina a strain of virus thought to belong here has been reported but has not yet been fully described.

Induced disease: In beet, clearing of veins, leaf curling, sharp protuberances from veins on lower surface of leaves, increase in number of rootlets, phloem degeneration followed by formation of supernumerary sieve tubes, retardation of growth. In tomato, (western yellow blight or tomato yellows), phloem degeneration followed by formation of supernumerary sieve tubes, retardation of growth, dropping of flowers and buds, rolling, yellowing and thickening of leaves, root decay, usually followed by death, sometimes by recovery. Occasionally there is relapse after recovery. In cucurbitaceous plants, stunting, bending upward of tip of runner, yellowing of old leaves, abnormally deep green in tip leaves and stem; Marblehead squash is tolerant, showing only mild witches' broom formation and phyllody. In bean, infected when young, thickening and downward curling of first trifoliate leaf, which becomes brittle and will break easily from the stem; leaves become chlorotic, plant stops growing and usually dies soon; older plants are also susceptible to infection; they may survive until

the end of the season, showing puckering and downward curling of leaves at the top of the plant, reduction in size of new leaves, and shortened internodes, or they may gradually become chlorotic and die.

Transmission: By leafhopper, *Eutettix tenellus* (Baker) (*CICADELLIDAE*) with 4 to 12 hour preinfective period. Through dodder, *Cuscuta campestris* Yuncker (*CONVOLVULACEAE*). Not, with any regularity at least, by mechanical inoculation of expressed juice. Not through seeds of diseased plants to seedlings germinating from them. The leafhopper, *Agalliana ensigera* Oman (*CICADELLIDAE*), is said to transmit a South American strain of sugar-beet curly-top virus, but evidence for identity of the virus has not yet been reported in detail.

Thermal inactivation: At 75° to 80° C in 10 minutes.

Filterability: Passes Berkefeld V, N, and W, Mandler medium and fine, and Chamberland L_1, L_2, L_5, L_7, L_9, L_{11} and L_{13} filters.

Other properties: Withstands alcohol and acetone treatments. A pH of 2.9 or lower inactivates, but an alkaline reaction as high as pH 9.1 does not inactivate, in 2 hours. Virus active after at least 8 years in tissues of thoroughly dried young sugar-beet plants, 6 months in dried leafhoppers, and 10 months in dried phloem exudate.

Strains: In general it has proved possible to modify strains by host passage, some hosts like *Chenopodium murale* L. appearing to select less virulent strains, others like *Stellaria media* (L.) Cyr. reversing this selection and restoring virulence.

Literature: Bennett, Jour. Agr. Res., *48*, 1934, 665–701; *50*, 1935, 211–241; *56*, 1938, 31–52; Phytopath., *32*, 1942, 826–827; Carsner, Phytopath., *15*, 1925, 745–757; U. S. Dept. Agr., Tech. Bull. 360, 1933; Jour. Agr. Res., *33*, 1926, 345–348; Dana, Phytopath., *28*, 1938, 649–656; Fawcett, Revista Industrial y Agricola de Tucumán, *16*, 1925, 39–46; Fife, Phytopath., *30*, 1940, 433–437; Giddings, Phytopath., *27*, 1937, 773–779; Jour. Agr. Res., *56*, 1938, 883–894; Lackey, Jour. Agr. Res., *55*, 1937, 453–460; Lesley and Wallace, Phytopath., *28*, 1938, 548–553; Murphy, *ibid.*, *30*, 1940, 779–784; Severin, Hilgardia, *3*, 1929, 595–636; Severin and Freitag, *ibid.*, *8*, 1933, 1–48; Severin and Henderson, Hilgardia, *3*, 1928, 339–393; Severin and Swezy, Phytopath., *18*, 1928, 681–690; Shaw, U. S. Dept. Agr., Bull. 181, 1910.

FAMILY V. SAVOIACEAE HOLMES.

(Handb. Phytopath. Viruses, 1939, 131.)

Viruses of the Savoy-Disease Group, causing diseases characterized mainly by crinkling of foliage. Vectors, true bugs (*PIESMIDAE* and *MIRIDAE*). There is a single genus.

Genus I. Savoia *Holmes.*

(*Loc. cit.*, 131.)

Characters those of the family. Generic name from French *chou de Savoie*, cabbage of Savoy, a cabbage with wrinkled and curled leaves.

The type species is *Savoia betae* Holmes.

Key to the species of genus Savoia.

I. Infecting beet.

1. *Savoia betae.*
2. *Savoia piesmae.*

II. Infecting rape and rutabaga.

3. *Savoia napi.*

1. **Savoia betae** Holmes. (Handb. Phytopath. Viruses, 1939, 132.) From Latin *beta*, beet.

Common names: Beet-Kräuselkrankheit virus, sugar-beet leaf-curl virus, sugar-beet leaf-crinkle virus, Kopfsalat virus.

Host: *CHENOPODIACEAE—Beta vulgaris* L., beet.

Geographical distribution: Germany, Poland.

Induced disease: In beet, veins of leaves swollen, retarded in growth, causing crinkling. New leaves remain small and incurved, forming a compact head. Old leaves die; plant succumbs before harvest time. Prepatent period in plant, 3 to 9 weeks.

Transmission: By tingid bug, *Piesma quadrata* Fieb. (*PIESMIDAE*). Not by inoculation of expressed juice.

Literature: Wille, Arb. Biol. Reichsanst. Land- u. Forstw., *16*, 1928, 115–167.

2. **Savoia piesmae** H. (*loc. cit.*, 132). From New Latin *Piesma*, generic name of insect vector.

Common name: Beet-savoy virus.

Host: *CHENOPODIACEAE—Beta vulgaris* L., beet.

Geographical distribution: United States (Michigan, Ohio, Minnesota, Nebraska, South Dakota, Colorado, Wyoming) and Canada.

Induced disease: In beet, leaves dwarfed, curled down, small veins thickened. Phloem necrosis in roots. Prodromal period in plant, 3 to 4 weeks.

Transmission: By tingid bug, *Piesma cinerea* (*PIESMIDAE*). Not by inoculation of expressed juice.

Literature: Coons et al., Phytopath., *27*, 1937, 125 (Abst.); Hildebrand and Koch, *ibid.*, *32*, 1942, 328–331.

3. **Savoia napi** H. (*loc. cit.*, 133). From New Latin *Napus*, former generic name of rape, *Brassica napus* L.

Common name: Rape-savoy virus.

Hosts: *CRUCIFERAE—Brassica napus* L., rape; *B. napobrassica* Mill., rutabaga.

Geographical distribution: Germany.

Induced disease: In rape, twisting and crinkling of young leaves; premature death of old leaves and of plants; in surviving plants, inhibition of growth in spring. In rutabaga, mottling and crinkling of leaves, with formation of fissures at leaf edges. Plants rarely killed.

Transmission: By inoculation of expressed juice. By the tarnished plant bug, *Lygus pratensis*Linn. (*MIRIDAE*). The insect vector retains this virus during intervals between crops.

Literature: Kaufmann, Arb. Biol. Reichsanst. Land- u. Forstw., *21*, 1936, 605–623; Mitteil. Landwirtsch., *37*, 1936; Pape, Deutsch. Landwirtsch. Presse, *26*, 1935.

FAMILY VI. LETHACEAE HOLMES.

(Handb. Phytopath. Viruses, 1939, 135.)

Virus strains of the Spotted-Wilt Group, causing diseases characterized by bronzing of foliage, streaking of stems, blighting of tips, necrotic spotting of foliage. Hosts, higher plants; vectors, thrips (*THRIPIDAE*). There is a single genus.

Genus I. Lethum *Holmes.*

(*Loc. cit.*, 135.)

Characters those of the family. Generic name from Latin *lethum*, death. At present there is but one known species, though this is reported to be nearly world-wide in distribution. In some areas it may have been confused with entities needing separate recognition.

The type species is *Lethum australiense* Holmes.

1. **Lethum australiense** Holmes (*loc. cit.*, 136). From Australia, where virus was first described.

Common names: Tomato spotted-wilt virus, kromnek or Kat River disease virus. Also, pineapple yellow-spot or side-rot virus (according to Sakimura, Phytopath., *30*, 1940, 281–299).

Hosts: Very numerous species in many families of higher plants. Among those most often noted are: *SOLANACEAE—Lycopersicon esculentum* Mill., tomato; *Nicotiana tabacum* L., tobacco; *Solanum tuberosum* L., potato. *COMPOSITAE—Lactuca sativa* L., lettuce. *LEGUMINOSAE—Pisum sativum* L., pea. *BROMELIACEAE—Ananas comosus* Merr., pineapple.

Geographical distribution: Australia, British Isles, United States, South Africa Hawaii, New Zealand, Europe, China, South America.

Induced disease: In tomato, bronze ring-like secondary lesions, plant stunted, some necrosis; later yellowish mosaic with some leaf distortion. Fruit frequently marked with concentric rings of pale red, yellow, or white. In tobacco, primary necrotic lesions followed by systemic necrosis, with stem streak, crook-neck, often stunting with subsequent wilting and death, sometimes temporary recovery followed by recurrence of systemic necrosis. In lettuce, plant yellowed, retarded in growth; brown blemishes in central leaves, affected spots dying, becoming like parchment but with brown margins. Axillary shoots may show chlorotic mottling. In pea, purplish necrotic streaks on stem; at first, leaves mottled; later, necrotic spots damage foliage; pods show circular necrotic spots or wavy lines, or, if severely affected, may collapse; seeds may show necrotic lesions. In potato, zonate necrotic spots on upper leaves, necrotic streaks on stems; stems collapse at top; plant is stunted, yield of tubers small. In pineapple, at first an initial spot or primary lesion $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter, raised, yellowish, on upper surface of young leaf; later chlorotic spotting of young leaves, crook-neck because of necrotic foci in stems and fruits (side rot); plant may die.

Transmission: By inoculation of expressed juice; the addition of fine carborundum powder to inoculum facilitates transmission by rubbing methods. By thrips, *Frankliniella lycopersici* Andrewartha (formerly identified as *F. insularis* Franklin), *F. occidentalis* Perg., *F. moultoni* Hood. and *F. schultzei* (Trybom) (*THRIPIDAE*). Also by *Thrips tabaci* Lind. (*THRIPIDAE*). In *F. lycopersici*, thrips must pick up virus while still a nymph; virus persists through pupation and emergence as adult; preinfective period in vector, 5 to 9 days. Virus is not transmitted through eggs of

infective thrips. Probably not through seeds of infected plants. Not through soil.

Immunological relationships: Infects tobacco plants previously infected with tobacco-mosaic, potato-mottle, tobacco-ringspot, and tomato-ringspot viruses.

Thermal inactivation: At 42° C in 10 minutes.

Filterability: Passes Gradocol membrane of 0.45 micron pore diameter.

Other properties: Virus readily inactivated by desiccation or by action of oxidizing agents; activity prolonged by presence of sodium sulfite, cystein, or by low temperatures. Unstable at pH values below 6 and above 9.

Literature: Ainsworth et al., Ann. Appl. Biol., *21*, 1934, 566–580; Andrewartha, Trans. Roy. Soc. of So. Australia, *61*, 1937, 163–165; Bald and Samuel, *ibid.*, *21*, 1934, 179–190; Berkeley, Scientific Agr., *15*, 1935, 387–392; Best, Austral. Chem. Inst. Jour. and Proc., *4*, 1937, 375–392; Best and Samuel, Ann. Appl. Biol., *23*, 1936, 509–537; 759–780; Carter, Phytopath., *29*, 1939, 285–287; Lewcock, Queensland Agr. Jour., *48*, 1937, 665–672; Linford, *ibid.*, *22*, 1932, 301–324; Magee, Agr. Gaz. of New South Wales, *47*, 1936, 99–100, 118; McWhorter and Milbrath, Phytopath., *25*, 1935, 897–898 (Abst.); Oregon Agr. Exp. Sta., Circ. *128*, 1938; Milbrath, Phytopath., *29*, 1939, 156–168; Moore, Nature, *147*, 1941, 480–481; Moore and Anderssen, Union of So. Africa, Dept. Agr., Science Bull. 182, 1939; Parris, Phytopath., *30*, 1940, 299–312; Rawlins and Tompkins, *ibid.*, *26*, 1936, 578–587; Sakimura, *ibid.*, *30*, 1940, 281–299; Samuel and Bald, Ann. Appl. Biol., *20*, 1933, 70–99; Jour. Agr. So. Australia, *37*, 1933, 190–195; Samuel et al., Counc. Scient. Indus. Res., Austral., Bull. 44, 1930; Ann. Appl. Biol., *22*, 1935, 508–524; Shapovalov, Phytopath., *24*, 1934, 1149 (Abst.); Smith, Nature, *127*, 1931, 852–853; Ann. Appl. Biol., *19*, 1932, 305–330; Jour. Minist. Agr., *39*, 1933, 1097–1104; Jour. Roy. Hort. Soc., *60*, 1935, 304–310; Snyder and Thomas, Hilgardia, *10*, 1936, 257–262; Takahashi and Rawlins, Phytopath., *24*, 1934, 1111–1115; Taylor and Chamberlain, New Zealand Jour. Agr., *54*, 1937, 278–283; Whipple, Phytopath., *26*, 1936, 918–920.

Strains: A strain differing somewhat from the type, var. *typicum* H. (*loc. cit.*, 136), has been described as damaging tomatoes in the northwestern United States. It has been given a distinctive varietal name:

1a. *Lethum australiense* var. *lethale* H. (*loc. cit.*, 138). From Latin *lethalis*, deadly. Common names: Tip-blight strain of tomato spotted-wilt virus, Oregon tip-blight virus, tomato die-back streak virus, tomato tip-blight virus. Differs from the type in causing necrotic leaf spotting, stem streaking, and tip blighting in most hosts, without mottling or bronzing of foliage; yet in *Tropaeolum majus* L., there is little necrosis. In tomato, systemic necrosis, terminal shoots blighted and blackened; dead tips stand upright above living foliage. Fruits rough and pitted, with internal pockets of necrotic tissue or with subepidermal necrosis, appearing externally as concentric brown bands. (McWhorter and Milbrath, Oregon Agr. Exp. Sta., Circ. *128*, 1938; Milbrath, Phytopath., *29*, 1939, 156–168.)

SUBORDER III. Zoophagineae *subordo novus.*

From Greek *phagein*, to eat, and *zoon*, an animal. Viruses infecting animals but having no plant hosts, so far as known.

Key to the families of suborder Zoophaginae.

1. Inducing diseases in insects as exclusive hosts.
   Family I. *Borrelinaceae*, p. 1225.
2. Inducing diseases of the pox group.
   Family II. *Borreliotaceae*, p. 1229.
3. Inducing diseases of the encephalitis group.
   Family III. *Erronaceae*, p. 1248.
4. Inducing diseases of the yellow-fever group.
   Family IV. *Charonaceae*, p. 1265.
5. Inducing diseases of the infectious anemia group.
   Family V *Trifuraceae*, p. 1282.
6. Inducing diseases of the mumps group.
   Family VI. *Rabulaceae*, p. 1284.

FAMILY I. BORRELINACEAE *FAM. NOV.*

Viruses causing polyhedral, wilt, and other diseases in arthropods. The genus *Borrelina* Paillot was originally spelled *Borrellina* by error; from Borrel, name of French scientist.

Key to the genera of family Borrelinaceae.

I. Known only as attacking lepidopterous insects.
   Genus I. *Borrelina*, p. 1225.
II. Known only as attacking the honey bee, a hymenopterous insect.
   Genus II. *Morator*, p. 1227.

Genus I. Borrelina *Paillot.*

(Compt. rend. Acad. Sci., Paris, *182*, 1926, 182.)

Viruses inducing polyhedral, wilt, and other diseases; hosts, Lepidoptera, so far as known.

The type species is *Borrelina bombycis* Paillot.

Key to the species of genus Borrelina.

I. Attacking silkworm.
   1. *Borrelina bombycis.*
II. Attacking nun moth.
   2. *Borrelina efficiens.*
III. Attacking gypsy moth.
   3. *Borrelina reprimens.*
IV. Attacking cabbage worm.
   4. *Borrelina brassicae.*
   5. *Borrelina pieris.*

1. **Borrelina bombycis** Paillot. (Compt. rend. Acad. Sci., Paris, *182*, 1926, 182.) From Latin *bombyx*, silkworm. (Note: Coccus-like bodies surrounded by non-staining substances, associated with the induced disease, received the provisional name *Chlamydozoon bombycis* from Prowazek, Arch. f. Protistenkunde, *10*, 1907, 363.)

Common names: Silkworm-jaundice virus, silkworm-grasserie virus, silkworm wilt virus, Gelbsucht virus, Fettsucht virus.

Host: *BOMBYCIDAE—Bombyx mori* (L.), silkworm.

Geographical distribution: Japan, Italy, France.

Induced disease: In silkworm, after prodromal period of 5 days or more, yellow spots on skin, polyhedral bodies in blood, inactivity, loss of appetite, irritability, weakening of body facilitating rupture from mechanical stress, eventual death.

Transmission: By feeding. Experimentally, also by injection.

Serological relationships: Specific agglutination, precipitation, and complement fixation.

Thermal inactivation: At 60° C in 15 to 20 minutes in blood.

Filterability: Passes Berkefeld N and V, Chamberland L_1, L_2, and L_3 filters.

Other properties: May survive at least 2 years in dry state. Stable between pH 5 and about pH 9. Sedimentation constant 17 S.

Literature: Aoki and Chigasaki, Cent. f. Bakt., I Abt., Orig., *86*, 1921, 481–485; Glaser and Lacaillade, Am. Jour. Hyg., *20*, 1934, 454–464; Glaser and Stanley, Jour. Exp. Med., *77*, 1943, 451–466; v. Prowazek, Cent. f. Bakt., I Abt., Orig., *67*, 1912, 268–284; Suzuki, Bull. Imperial Kyoto Sericultural College, *1*, 1929, 45–75; Trager, Jour. Exp. Med., *61*, 1935, 501–513.

2. **Borrelina efficiens** *spec. nov.* From Latin *efficiens*, effective, in reference to effectiveness of this virus in controlling nun-moth infestations.

Common names: Nun-moth disease virus, nun-moth wilt virus, Wipfelkrankheit virus.

Host: *LYMANTRIIDAE—Lymantria monacha* (L.), nun moth.

Geographical distribution: Europe.

Induced disease: In eggs, larvae, pupae and occasionally adults of nun moth, polyhedral bodies in affected tissues. Blood of sick larvae turbid; later, blood cells few; contents of body finally become a gray-brown, semifluid mass.

Transmission: By feeding.

Thermal inactivation: At 55 to 60° C in 5 to 10 minutes.

Filterability: Fails to pass Berkefeld and Chamberland filters.

Other properties: May remain viable at least 2 years in dry state.

Literature: Escherich and Miyajima, Naturwissens. Ztschr. f. Forst- u. Landwirtschaft, *9*, 1911, 381–402; Wachtl and Kornauth, Mitth. a. d. forstl. Versuchswesen Österreichs, *16*, 1893, 1–38; Wahl, Centralbl. Gesam. Forstw., *35*, 1909, 164–172; 212–215; *36*, 1910, 377–397; *37*, 1911, 247–268; *38*, 1912, 355–378.

3. **Borrelina reprimens** *spec. nov.* From Latin *reprimere*, to restrain.

Common name: Gypsy-moth wilt virus.

Host: *LYMANTRIIDAE—Porthetria dispar* (L.), gypsy moth.

Geographical distribution: United States.

Induced disease: In gypsy moth caterpillar, flaccidity, disintegration of tissues, eventual collapse as a watery sack. Death occurs in 13 to 29 (average 21) days after infection; caterpillar may remain attached to its support by prolegs; skin ruptures easily. Polyhedral bodies originate in nuclei of the tracheal matrix, hypodermal, fat, and blood cells.

Transmission: By feeding on contam-

inated foliage. Not through undamaged skin.

Filterability: Passes Berkefeld N, not Pasteur-Chamberland F, filter.

Literature: Chapman and Glaser, Jour. Econ. Entomol., *8*, 1915, 140–150; *9*, 1916, 149–167; Glaser, Jour. Agr. Res., *4*, 1915, 101–128; Science, *48*, 1918, 301–302; Glaser and Chapman, Jour. Econ. Entomol., *6*, 1913, 479–488.

4. **Borrelina brassicae** Paillot. (Compt. rend. Acad. Sci., Paris, *182*, 1926, 182.) From name of host, *Pieris brassicae*.

Common name: Cabbage-worm grasserie virus.

Host: *PIERIDAE—Pieris brassicae* (L.), cabbage worm.

Induced disease: In cabbage worm, no nuclear or cytoplasmic inclusions; nuclei of fat and hypodermal cells hypertrophied and soon disorganized.

Transmission: By feeding.

Other properties: Described as submicroscopic in size, intracytoplasmic.

Literature: Paillot, *loc. cit.*; Ann. Inst. Pasteur, *40*, 1926, 314–452; L'infection chez les insectes. Immunité et symbiose, 535 pages, Trévoux, Patissier, 1933.

5. **Borrelina pieris** Paillot. (Compt. rend. Acad. Sci., Paris, *182*, 1926, 182.) From New Latin *Pieris*, generic name of host.

Common name: Virus of nuclear disease of pierids.

Host: *PIERIDAE—Pieris brassicae* (L.), cabbage worm.

Induced disease: In cabbage worm, body yellowish below, tears easily just before death; chromatin of nuclei in fat and blood cells condensed in irregular masses; cytoplasmic inclusions staining faintly red in Giemsa preparations.

Transmission: By feeding.

Other properties: Described as intracytoplasmic, less than 0.1 micron in diameter.

Literature: Paillot, *loc. cit.*; Ann. Inst. Pasteur, *40*, 1926, 314–452; L'infection chez les insectes. Immunité et symbiose, 535 pages, Trévoux, Patissier, 1933.

Appendix: *Borrelina flacheriae* quoted from Paillot, L'infection chez les insects. 535 pp., Trevoux, Patissier, 1935, see p. 96. Cause of gattine in the silkworm, *Bombyx mori* L. No previous reference to a description of this species has been found.

Genus II. **Morator** *gen. nov.*

Only one species at present, inducing the disease known as sacbrood of the honey bee. Generic name from Latin *morator*, loiterer. The type, and only, species is *Morator aetatulae spec. nov.*

1. **Morator aetatulae** *spec. nov.* From Latin *aetatula*, early period of life, in reference to attack on immature stages of host, exclusively.

Common name: Honey-bee sacbrood virus.

Host: *APIDAE—Apis mellifera* L., honey bee (immature stages only).

Insusceptible species: *LYMANTRIIDAE—Porthetria dispar* (L.), gypsy moth.

Geographical distribution: United States.

Induced disease: In the honey bee, immature stages only are susceptible; infected larvae die, usually after capping, some of the dead brood being uncapped by the bees. Occasionally caps are punctured. Affected areas of comb are usually small and scattered. Each larva is extended along its cell, head turned upward toward the roof. A larva recently dead appears light yellow, light gray, or light brown, soon darkening to brown or almost black. Cuticle of dead larva tough, permitting extraction of the sac-

like mass without rupture; contents watery with many suspended, fine, brown particles. There are no characteristic intracellular bodies in affected tissues. Dead larvae eventually dry down to form scales that are black and roughened, that separate readily from the cell wall, and that may be lifted out intact. Colonies tend to lose virus spontaneously.

Transmission: By contamination of food. Not by hands, clothing, or tools. Perhaps through water supply of insects.

Thermal inactivation: In water, at 58° C in 10 minutes. In honey, at 70 to 73° C in 10 minutes.

Filterability: Passes Berkefeld and Pasteur-Chamberland filters.

Other properties: Withstands drying 20, not 22, days, exposure to sunlight 7 hours or less, storage in honey a month or more, ½ to 2 per cent aqueous solutions of carbolic acid 3 weeks or more.

Literature: McCray and White, U. S. Dept. Agr., Dept. Bull. 671, 1918; White, U. S. Dept. Agr., Bur. of Entomol., Circ. 169, 1913; U. S. Dept. Agr., Dept. Bull. 92, 1914; *ibid.*, Dept. Bull. 431, 1917.

FAMILY II. BORRELIOTACEAE *FAM. NOV.*

Viruses of the Pox Group, inducing diseases characterized in general by discrete primary and secondary lesions of the nature of macules, papules, vesicles, or pustules.

Key to the genera of family **Borreliotaceae.**

I. Viruses of the Typical Pox-Disease Group.
Genus I. *Borreliota*, p. 1229.
II. Viruses of the Varicella Group.
Genus II. *Briareus*, p. 1233.
III. Viruses of the Herpes Group.
Genus III. *Scelus*, p. 1234.
IV. Viruses of the Foot-and-Mouth-Disease Group.
Genus IV. *Hostis*, p. 1239.
V. Viruses of the Wart-Disease Group.
Genus V. *Molitor*, p. 1240.

Genus I. Borreliota *Goodpasture.*

(Science, *77*, 1933, 121.)

Viruses of the Typical Pox-Disease Group, inducing diseases characterized by formation of papules, pustules, and scabs, shed with or without scarring. Generic name from *Borrel*, investigator who first discovered the specific granules of fowl pox and Latinized name of the smallest Greek letter, *iota*, signifying smallest particle. The name *Cytoryctes variolae* Guarnieri 1892 was based on intracellular inclusions, Guarnieri bodies, as supposed sporozoan parasites (Calkins, Jour. Med. Res., *11*, 1904, 136–172).

The type species is *Borreliota avium* Goodpasture.

Key to the species of genus **Borreliota.**

I. Affecting domestic fowl.
1. *Borreliota avium.*
II. Affecting man principally, although strains have become adapted to cow, rabbit, etc.
2. *Borreliota variolae.*
III. Affecting swine.
3. *Borreliota suis.*

1. **Borreliota avium** (Lipschütz) Goodpasture. (*Strongyloplasma avium* Lipschütz, in Kolle, Kraus and Uhlenhuth, Handbuch der pathogenen Mikroorganismen, 3 Auft., *8*, 1930, 314; Goodpasture, Science, *77*, 1933, 121.) From Latin *aves*, fowl of the air.

Common names: Fowl-pox virus; also known as poultry-pox virus, chicken-pox virus (but not the virus of the same name attacking man rather than the chicken), or virus of epithelioma contagiosum of fowls; strains have been studied under the names Kikuth's canary virus and pigeon-pox virus.

Hosts: Chicken, turkey, pigeon, goose, duck, guinea fowl, quail, hawk, pheasant, partridge, bunting sparrow, canary. Experimentally, also English sparrow, chick embryo.

Insusceptible species: Man, goat, sheep, mouse, rat, guinea pig.

Geographical distribution: Europe, Asia, North America; perhaps coextensive with the area in which chickens are grown under conditions of domestication.

Induced disease: In chicken, hyperplastic nodular lesions of the skin, diphtheritic membranes in mouth and throat, discharges from eyes and nose; nodules eventually dry up and fall off, usually without leaving scars. Inclusion bodies, known as Bollinger bodies, believed to represent aggregates of minute Borrel bodies or virus particles, leave much grayish-white ash when incinerated; break readily after digestion by 1 per cent trypsin in 0.2 per cent sodium bicarbonate. Borrel bodies coccoid, 0.25 microns in diameter. On chorioallantoic membrane of chick embryo, proliferation and hyperplasia, or necrosis.

Transmission: By contact, perhaps through wound infection. By blood-sucking dipterous insects. Experimentally, by scarification of skin or buccal mucosa; by intravenous, intradermal, subcutaneous, intramuscular, or intraperitoneal inoculation. May be passed in series by nasal instillation in chickens, obvious mucosal changes occurring only occasionally. Experimentally, by mosquitoes (*CULICIDAE*), *Aëdes aegypti* L., *A. stimulans* Walker, *A. vexans* Meigen (as long as 27 days from time of feeding on infective material), and *Culex pipiens* L. (indefinitely after infective feeding, as long as the individual mosquito lives); in *C. pipiens*, the virus has been found also under natural conditions.

Serological relationships: Neutralizing and elementary-body-agglutinating antisera specific. Antivaccinial serum from rabbit ineffective against fowl-pox virus, although neutralizing vaccinia virus.

Immunological relationships: No cross immunity with respect to vaccinia virus in the chicken.

Thermal inactivation: At 60° C in 8 minutes; at 56° C in 30 minutes.

Filterability: Passes Berkefeld V, not Chamberland L_2, filter candle.

Other properties: Drying at room temperature *in vacuo* does not inactivate. Viable after storage at least 24 months at 0 to 4° C, dry.

Strains: A strain known as Kikuth's canary virus has been studied in some detail. When introduced into the rabbit it induces formation of neutralizing antibodies that react strongly with homologous virus, moderately against fowl-pox virus. Antivaccinial serum is ineffective against it. In canaries, it induces proliferation of dermal epithelium with cytoplasmic inclusions, the inflammatory process being characterized by predominantly mononuclear cells with vacuolated cytoplasm; in the lung there is massive accumulation of large mononuclear cells containing the specific cytoplasmic inclusions; the disease is regularly fatal. Passes Berkefeld N filter. Size estimated as 120 millimicrons by centrifugation. (Bechhold and Schlesinger, Ztschr. f. Hyg., *115*, 1933, 354–357; Burnet, Jour. Path. and Bact., *37*, 1933, 107–122; Burnet and Lush, Brit. Jour. Exp. Path., *17*, 1936, 302–307; Gaede, Cent. f. Bakt., I Abt., Orig., *135*, 1935, 342–346; Kikuth and Gollub, *ibid.*, *125*, 1932, 313–320.)

Literature: Andervont, Am. Jour. Hyg., *6*, 1926, 719–754; Brandly and Dunlap, Jour. Am. Vet. Med. Assoc., *95*, 1939, 340–349; Brandly et al., Am. Jour. Vet. Res., *2*, 1941, 190–192; Brody, Cornell Agr. Exp. Sta. (Ithaca). Memoir 195, 1936; Buddingh, Jour. Exp. Med., *67*, 1938, 933–940; Burnet and Lush, Brit. Jour. Exp. Path., *17*, 1936, 302–307; Danks, Am. Jour. Path., *8*, 1932, 711–716; Findlay, Proc. Roy. Soc. London, Ser. B, *102*, 1928, 354–379; Goodpasture and A. M. Woodruff, Am. Jour. Path., *6*, 1930, 699–712; Goodpasture and C. E. Woodruff, *ibid.*, *7*, 1931, 1–8; Irons, Am. Jour. Hyg., *20*, 1934, 329–351; Kligler and Ashner, Proc. Soc. Exp. Biol. and Med., *28*, 1931, 463–465; Kligler et al., Jour. Exp. Med., *49*, 1929, 649–660; Ledingham, Lancet, *221*, 1931 (*2*), 525–526; Ludford and Findlay, Brit. Jour. Exp. Path., *7* 1926, 256–264; Matheson et al, Poultry Science, *10*, 1931, 211–223; Megrail, Am. Jour. Hyg., *9*, 1929, 462–465; Nelson, Jour. Exp. Med., *74*, 1941, 203–212; A. M. Woodruff, and Goodpasture, Am. Jour.

Path., *7*, 1931, 209–222; C. E. Woodruff, *ibid.*, *6*, 1930, 169–174; C. E. Woodruff, and Goodpasture, *ibid.*, *5*, 1929, 1–10; *6*, 1930, 713–720.

2. Borreliota variolae (Lipschütz) Goodpasture. (*Strongyloplasma variolae* Lipschütz, in Kolle, Kraus and Uhlenhuth, Handbuch der pathogenen Mikroorganismen, 3 Aufl., *8*, 1930, 317; Goodpasture, Science, *77*, 1933, 121.) From New Latin *variola*, smallpox.

Common names: Variola virus, smallpox virus. Most studies of this virus have been concerned with the vaccinia strain; see Strains below.

Hosts: Man, cow and rabbit are susceptible to strains that appear especially adapted to them (see *Strains* below). Experimentally, also chicken (and chick embryo); *Chrysemys marginata*, turtle; guinea pig, horse, pig; *Macaca mulatta* (Zimmermann), rhesus monkey; *M. irus*, cynomolgus monkey; orang-outang; *Macacus fuscatus*.

Geographical distribution: Nearly world-wide, except where excluded by isolation or protective vaccination.

Induced disease: In man, mild to severe smallpox, sometimes with pocks few and discrete but often with pocks numerous and coalescing; onset sudden, 6 to 22 days (average 12) after infection; headache, vomiting, fever, often rashes on body before appearance of the specific eruption, bright red spots becoming vesicular and eventually pustular; the pocks are commonest on face, forearms, wrists, palms of hands, and soles of feet; pustules gradually become flattened scabs and drop off, leaving no scar if superficial and not secondarily infected; in hemorrhagic smallpox there are numerous hemorrhages into the skin and mortality is high, death often preceding formation of pustules; severity of disease and mortality roughly proportional to the amount of eruption on the face.

Transmission: By contact with infected individuals or contaminated articles; perhaps by droplet infection, obvious primary lesions characterizing experimental transmission by scarification but not natural spread.

Serological relationships: Hyperimmune calf serum neutralizes virus. Neutralization depends on an antibody not involved in agglutination and precipitation. Antivaccinial serum gives complement fixation in the presence of variola virus. One agglutinogen (L) labile at 56° C, one (S) stable at 95° C; both are parts of a single protein but can be degraded independently; chymotrypsin destroys activity of S, not L. Increasing neutralization in immune serum and virus mixtures *in vitro* with progressive incubation; partial reactivation on simple dilution. Antivaccinial sera agglutinate Paschen bodies of vaccinia but not Borrel bodies of fowl pox; anti-fowl-pox sera agglutinate Borrel but not Paschen bodies. No cross reactions with herpes virus.

Immunological relationships: In vaccinia-immune swine, protective substances pass *via* colostrum, conveying passive immunity to young for 2 to 3 months after birth. In man, immunity against variola virus is conferred by earlier infection with vaccinia strain. In hen, previous infection with fowl-pox virus does not immunize with respect to vaccinia virus.

Thermal inactivation: At 55° C in 20 minutes.

Filterability: Passes Berkefeld V, not Mandler, filter.

Other properties: Density about 1.16. Sedimentation constant 5000×10^{-13} (corrected to water at 20° C). Retains activity in glycerine best at pH 7.0. 0.1 per cent gelatin delays spontaneous inactivation at 5 to 10° C. Withstands absolute alcohol, ether, acetone, and petroleum ether 1 hour in dry samples at 4° C without decrease in activity. Inactivated without disruption by sonic vibrations of about 8900 cycles per second. Diameter estimated as 125 to 175 millimicrons by filtration; 236 to 252 millimicrons by ultracentrifugation. Electron micrographs show limiting surface

membrane, dense granules (usually 5) within; tendency to rectangular outlines with rounded corners. At least 5.6 per cent of virus is reported to be thymonucleic acid. Contains nitrogen, 15.3 per cent; carbon, 33.7 per cent; phosphorus, 0.57 per cent; phospholipid (lecithin), 2.2 per cent; neutral fat, 2.2 per cent; reducing sugars after hydrolysis, 2.8 per cent; cystine, 1.9 per cent; copper, 0.05 per cent.

Strains: Besides the typical variola strain, var. *hominis* Goodpasture (Science, *77*, 1933, 121), several distinctive strains have been studied. A spontaneous cowpox strain differs in some antigens but affords cross immunity with respect to var. *bovis* Goodpasture (*loc. cit.*, 121), vaccinia virus, which in turn immunizes against typical variola virus. A spontaneous rabbit-pox strain, serologically resembling neurovaccine virus, is believed to exist independently in Europe and the United States. The varieties *equi* (horse-pox virus), *porci* (swine strain), and *ovium* (sheep and goat pox virus) have been attributed to this species by Goodpasture (*loc. cit.*, 121). The alastrim strain (causing *variola minor*) differs from the type in producing a relatively mild disease in man and in inducing the formation of a distinctive type of intracellular inclusion in affected tissues.

Literature: Amies, Jour. Path. and Bact., *47*, 1938, 205–222; Andervont, Am. Jour. Hyg., *7*, 1927, 804–810; Behrens and Ferguson, Jour. Inf. Dis., *56*, 1935, 84–88; Behrens and Nielson, *ibid.*, *56*, 1935, 41–48; Buddingh, Am. Jour. Hyg., *38*, 1943, 310–322; Craigie and Wishart, Brit. Jour. Exp. Path., *15*, 1934, 390–398; Jour. Exp. Med., *64*, 1936, 819–830; Dearing, Am. Jour. Hyg., *20*, 1934, 432–443; Douglas et al., Jour. Path. and Bact., *32*, 1929, 99–120; Downie, Brit. Jour. Exp. Path., *20*, 1939, 158–176; Eagles, *ibid.*, *16*, 1935, 181–188; Elford and Andrewes, Brit. Jour. Exp. Path., *13*, 1932, 36–42; Goodpasture, Woodruff, and Buddingh, Am. Jour. Path., *8*, 1932, 271–282; Green et al., Jour. Exp. Med., *75*, 1942, 651–656; Greene, *ibid.*, *61*, 1935, 807–831; Herzberg, Ztschr. Immunitätsforsch. u. exper. Therap., *86*, 1935, 417–441; Hoagland et al., Jour. Exp. Med., *71*, 1940, 737–750; *72*, 1940, 139–147; *74*, 1941, 69–80, 133–144; *76*, 1942, 163–173; Hu et al., Jour. Exp. Med., *63*, 1936, 353–378; Keogh, Jour. Path. and Bact., *43*, 1936, 441–454; Ledingham, Brit. Jour. Exp. Path., *5*, 1924, 332–349; Jour. Path. and Bact., *35*, 1932, 140–142; Macfarlane and Dolby, Brit. Jour. Exp. Path., *21*, 1940, 219–227; Macfarlane and Salaman, *ibid.*, *19*, 1938, 184–191; McFarlane et al., *ibid.*, *20*, 1939, 485–501; Moriyama, Arch. f. Virusforsch., *1*, 1940, 422–429; Nelson, Jour. Exp. Med., *60*, 1934, 287–291; *78*, 1943, 231–239; Nye and Parker, Am. Jour. Path., *5*, 1929, 147–155; Parker, Jour. Exp. Med., *67*, 1938, 361–367, 725–738; Parker and Muckenfuss, Jour. Infect. Dis., *53*, 1933, 44–54; Parker and Rivers, Jour. Exp. Med., *62*, 1935, 65–72; *64*, 1936, 439–452; *65*, 1937, 243–249; Paschen, Deutsch. med. Wchschr., *39*, 1913, 2132–2136; Pearce et al., Jour. Exp. Med., *63*, 1936, 241–258, 491–507; Jour. Path. and Bact., *43*, 1936, 299–312; Pickels and Smadel, Jour. Exp. Med., *68*, 1938, 583–606; Rhodes and van Rooyen, Jour. Path. and Bact., *44*, 1937, 357–363; Rivers and Ward, Jour. Exp. Med., *58*, 1933, 635–648; *62*, 1935, 549–560; Rivers et al., *ibid.*, *65*, 1937, 677–685; *69*, 1939, 857–866; Rosahn et al., Jour. Exp. Med., *63*, 1936, 259–276, 379–396; Rosenau and Andervont, Am. Jour. Hyg., *13*, 1931, 728–740; Salaman, Brit. Jour. Exp. Path., *18*, 1937, 245–258; Shedlovsky and Smadel, Jour. Exp. Med., *75*, 1942, 165–178; Smadel and Rivers, *ibid.*, *75*, 1942, 151–164; Smadel et al., *ibid.*, *68*, 1938, 607–627; *71*, 1940, 373–389; *77*, 1943, 165–171; Smith, Jour. Path. and Bact., *33*, 1930, 273–282; Sprunt, Proc. Soc. Exp. Biol. and Med., *51*, 1942, 226–227; Jour. Exp. Med., *75*, 1942, 297–304; Stritar and Hudson, Am. Jour. Path., *12*, 1936, 165–174; Ward, Jour. Exp. Med., *50*, 1929, 31–40.

3. Borreliota suis *spec. nov.* From Latin *sus*, swine.

Common name: Swine-pox virus (not

the vaccinia strain of variola virus in swine).

Host: *SUIDAE—Sus scrofa* L., domestic swine.

Insusceptible species: Rabbit.

Geographical distribution: United States (Iowa).

Induced disease: In swine, locally, reddened hyperemic papules 3 to 7 mm in diameter; papules become briefly vesicular, then change gradually to true pustules, finally forming dark brown to blackish scabs which are shed after a few weeks without scarring; no secondary lesions in hogs free from lice, but in infested animals numerous secondary lesions appear 1 to 2 weeks after primary lesions and are commonly most numerous in the inguinal and axillary regions. Mortality negligible but growth retarded. Virus has been recovered from hog louse after feeding on affected swine.

Transmission: By hog louse, *Haematopinus suis* (*HAEMATOPINIDAE*), probably mechanically. By experimental scarification of skin.

Serological relationships: No reaction with neutralizing sera specific for vaccinia virus.

Immunological relationships: Specific immunity in swine after attack, but no cross immunity with respect to vaccinia virus.

Filterability: Passes Berkefeld V and N filters.

Literature: Csontos and von Nyiredy, Deutsch. tierärztl. Wchnschr., *41*, 1933, 529–532; Schwarte and Biester, Am. Jour. Vet. Res., *2*, 1941, 136–140; Shope, Arch. f. Virusforsch., *1*, 1940, 457–467.

Genus II. **Briareus** *gen. nov.*

Viruses of the Varicella Group, causing diseases characterized by reddened spots and rings in affected tissues, becoming papular or vesicular. Generic name from Latin *Briareus*, name of a hundred-armed giant.

The type species is *Briareus varicellae spec. nov.*

Key to the species of genus **Briareus.**

I. Causing chicken pox and herpes zoster in man. 1. *Briareus varicellae.*

II. Causing measles in man. 2. *Briareus morbillorum.*

1. **Briareus varicellae** *spec. nov.* From New Latin *varicella*, chicken pox.

Common names: Varicella virus, chicken-pox virus; much evidence for identity with so-called herpes-zoster virus has been presented.

Host: *HOMINIDAE—Homo sapiens* L., man.

Insusceptible species: Chick embryo.

Geographical distribution: World-wide.

Induced disease: In man, usually abrupt onset, rash at first macular, soon papular and vesicular; vesicles generally discrete, soon rupturing, healing with scab formation and itching; separation of deeper scabs may leave persistent scars; in severe cases there may be stomatitis, laryngitis, and nasal lesions. In human skin grafted on chorioallantois of chick embryo, experimentally, pustular lesions as in natural disease, with intranuclear acidophilic inclusions; no gross vesiculation.

Transmission: By contact. By spread of droplets. Children in contact with herpes zoster patients sometimes contract varicella.

Serological relationships: Majority of herpes zoster sera that agglutinate zoster antigen also agglutinate elementary bodies of varicella; complement fixation tests also indicate relationship of virus from herpes zoster and varicella. Chicken-pox sera do not flocculate smallpox brain-virus antigen.

Immunological relationships: Children previously having varicella are immune to inoculation with herpes zoster virus.

Literature: Amies, Brit. Jour. Exp. Path., *15*, 1934, 314–320; Brain, *ibid.*, *14*, 1933, 67–73; Bruusgaard, Brit. Jour. Derm. Syph., *44*, 1932, 1–24; Goodpasture and Anderson, Am. Jour. Path., *20*, 1944, 447–453; Havens and Mayfield, Jour. Inf. Dis., *50*, 1932, 242–248; Irons et al., Am. Jour. Hyg., *55*, (B), 1941, 50–55; Kundratitz, Monatsschr. Kinderheilk., *29*, 1925, 516–523; Lipschütz and Kundratitz, Wien. klin. Woch., *38*, 1925, 499–503.

2. **Briareus morbillorum** *spec. nov.* From New Latin *morbilli*, measles.

Common name: Measles virus.

Host: *HOMINIDAE—Homo sapiens* L., man. Experimentally, also *CERCOPITHECIDAE—Macaca mulatta* (Zimmermann), rhesus monkey. *PHASIANIDAE—Gallus gallus* (L.), chick embryo (no lesions, but 30 serial passages).

Geographical distribution: World-wide except in isolated communities.

Induced disease: In man, after incubation period of 7 to 21 days, bright red spots on buccal mucosa, especially near first molar tooth (Koplik's spots) followed by rash on face, head, neck, then arms, trunk, and legs; papules often crescents, lesions usually discrete; rash fades, leaving brownish discoloration and desquamation.

Transmission: By contact. By droplets.

Serological relationships: Convalescent serum is reported to modify the course of the induced disease if administered intravenously in the preeruptive stage.

Immunological relationships: Specific immunity in man after attack.

Thermal inactivation: At 55° C in 15 minutes.

Filterability: Passes Berkefeld N filter candle and Seitz EK disks.

Other properties: Viable at −35° C for at least 4 weeks. Not inactivated by 10 per cent anesthetic ether in 40 minutes.

Literature: Blake and Trask, Jour. Exp. Med., *33*, 1921, 385–412; Gordon and Knighton, Am. Jour. Path., *17*, 1941, 165–176; Hedrich, Am. Jour. Hyg., *17*, 1933, 613–636; Kohn et al., Jour. Am. Med. Assoc., *111*, 1938, 2361–2364; Rake and Shaffer, Jour. Immunol., *38*, 1940, 177–200; Rake et al., Jour Inf. Dis., *69*, 1941, 65–69; Scott and Simon, Am. Jour. Hyg., *5*, 1925, 109–126.

Genus III. Scelus *gen. nov.*

Viruses of the Herpes Group, inducing diseases characterized in general by vesicular primary lesions, sometimes with subsequent involvement of the nervous system. Generic name from Latin *scelus*, rascal.

The type species is *Scelus recurrens spec. nov.*

Key to the species of genus **Scelus.**

I. In man, cause of so-called fever blisters, herpes febrilis.
   1. *Scelus recurrens.*

II. In swine, cause of pseudorabies.
   2. *Scelus suillum.*

III. In monkey.
   3. *Scelus beta.*

IV. In rabbit, course of the induced disease in nature unknown.
   4. *Scelus tertium.*

V. In sheep, cause of ovine balano-posthitis.
   5. *Scelus ulceris.*

VI. In mice, cause of ectromelia.
   6. *Scelus marmorans.*

VII. In cattle, cause of erosive stomatitis.
   7. *Scelus bovinum.*

1. **Scelus recurrens** *spec. nov.* From Latin *recurrere*, to recur. Note: The name *Neurocystis herpetii* Levaditi and Schoen (Compt. rend. Soc. Biol., Paris, *96*, 1927, 961) was applied provisionally to the causative microorganism of herpes, in the expectation that future research would show inclusion bodies in affected tissues to be stages in its life cycle.

Common names: Herpes virus, virus of herpes simplex, virus of herpes febrilis (not herpes zoster virus, for which see varicella virus), virus of keratitis dendritica, virus of aphthous stomatitis (of man).

Host: *HOMINIDAE—Homo sapiens* L., man. Experimentally, also rabbit, guinea pig, white mouse, cat, goose, hedgehog, and, though difficult to infect, dog and pigeon. Chick embryo (but not chicken). Also *CERCOPITHECIDAE —Cercocebus fuliginosus* E. Geoffrey, *Macacus cynomolgus*. *CEBIDAE—Cebus olivaceus*.

Insusceptible species: White rat; *Bufo viridis*; *Cercopithecus callithrix*; chicken (except embryo).

Geographical distribution: Probably world-wide.

Induced disease: In man, usually acquired in first three years of life, sometimes as aphthous stomatitis; virus probably retained often through life, sometimes with periodic reappearance of dermal lesions, which are vesicular and heal soon. In white mouse, by experimental inoculation of skin, small inflamed vesicular primary lesions about 5 days after inoculation, usually forming scabs and healing a few days later, but sometimes persisting; if on tail, followed by swelling and paralysis of tail, ascending paralysis and death, or by recovery with acquired immunity; if near head, followed by encephalitis and death; intraperitoneal and sometimes other inoculations immunize; relapse with recurrence of primary lesions rare. In chick embryo, white, opaque, circular or ring-like primary lesions of small size on chorioallantoic membrane, with or without necrotic secondary lesions in liver, heart, lungs, spleen, and kidneys; virus enters membrane 1 to 4 hours after it is dropped on its surface; primary lesions may be counted in 48 hours.

Transmission: By contacts. Experimentally, by skin scarification; in guinea pig, by feeding.

Serological relationships: Distant relationship to pseudorabies virus, *Scelus suillum*, shown by moderate protection against this virus conferred by some anti-herpes sera. No relationship to vaccinia virus or to virus III of rabbits demonstrable by neutralization tests. Specific complement fixation. Neutralizing antibody forms reversible union with virus, at least for a time, though with strong mixtures partial irreversibility finally occurs.

Immunological relationships: Formalinized virus and non-lethal strains of virus immunize specifically. No cross immunity with vaccinia virus.

Thermal inactivation: At 50 to 52° C in 30 minutes, when moist; at 90 to 100° C in 30 minutes, when dry. At 41.5° C in 50 to 80 hours.

Filterability: Passes Berkefeld V filter with slight loss.

Other properties: Diameter, by centrifugation, computed as 180 to 220 millimicrons; by filtration, 100 to 150 millimicrons. Specific gravity, 1.15. Inactivated by repeated freezing and thawing; also by pressure of 3000 atmospheres for 30 minutes. Viable dry at least 18 months at 4° C, in 50 per cent glycerine at least 6 months. Not inactivated at 4° C in 1 per cent aqueous gentian violet. Charged negatively in solutions of hydrogen-ion concentration up to about pH 8. Isoelectric point, pH 7.2 to 7.6. Inactivated by incubation *in vitro* at pH 6 with synthetic vitamin C (ascorbic acid).

Literature: Anderson, Science, *90*, 1939, 497; Am. Jour. Path., *16*, 1940, 137–156; Andervont, Jour. Inf. Dis., *44*, 1929, 383–393; *45*, 1929, 366–385; *49*, 1931, 507–529; Andrewes, Jour. Path. and Bact., *33*, 1930, 301–312; Bassett et al., Compt. rend. Acad. Sci., Paris, *200*, 1935, 1882–1884; Bechhold and Schlesinger, Ztschr.

f. Hyg., *115*, 1933, 342–353; Bedson, Brit. Jour. Exp. Path., *12*, 1931, 254–260; Bedson and Bland, *ibid.*, *9*, 1928, 174–178; Blanc and Caminopetros, Compt. rend. Soc. Biol., Paris, *84*, 1921, 859–860; Boak et al., Jour. Exp. Med., *71*, 1940, 169–173; Brain, Brit. Jour. Exp. Path., *13*, 1932, 166–171; Buggs and Green, Jour. Inf. Dis., *58*, 1936, 98–104; Burnet and Lush, Jour. Path. and Bact., *48*, 1939, 275–286; *49*, 1939, 241–259; Lancet, *236*, 1939 (*1*), 629–631; Burnet et al., Austral. Jour. Exp. Biol. and Med. Sci., *17*, 1939, 35–40; Dawson, Am. Jour. Path., *9*, 1933, 1–6; Elford et al., Jour. Path. and Bact., *36*, 1933, 49–54; Findlay and MacCallum, Lancet, *238*, 1940 (*1*), 259–261; Fischl and Schaefer, Klin. Wochnschr., *8*, 1929, 2139–2143; Flexner, Jour. Gen. Physiol., *8*, 1927, 713–726; Jour. Exp. Med., *47*, 1928, 9–36; Friedenwald, Arch. Ophthalmol., *52*, 1923, 105–131; Goodpasture, Medicine, *8*, 1929, 223–243; Goodpasture and Teague, Jour. Med. Res., *44*, 1923, 121–138; Gunderson, Arch. Opthalmol., *15*, 1936, 225–249; Holden, Jour. Inf. Dis., *50*, 1932, 218–236; Keddie and Epstein, Jour. Am. Med. Assoc., *117*, 1941, 1327–1330; Levaditi and Lepine, Compt. rend. Acad. Sci., Paris, *189*, 1929, 66–68; Levaditi and Nicolau, Compt. rend. Soc. Biol., Paris, *90*, 1924, 1372–1375; Long, Jour. Clin. Investigation, *12*, 1933, 1119–1125; Magrassi, Boll. Ist. Sieroterap. Milanese, *14*, 1935, 773–790; McKinley, Proc. Soc. Exp. Biol. and Med., *26*, 1928, 21–22; Naegeli and Zurukzoglu, Cent. f. Bakt. I Abt., Orig., *135*, 1935, 297–299; Nicolau and Kopciowska, Ann. Inst. Pasteur, *60*, 1938, 401–431; Parker and Nye, Am. Jour. Path., *1*, 1925, 337–340; Perdrau, Proc. Roy. Soc. London, Ser. B, *109*, 1931, 304–308; Jour. Path. and Bact., *47*, 1938, 447–455; Remlinger and Bailly, Comp. rend. Soc. Biol., Paris, *94*, 1926, 734–736; 1064–1066; *95*, 1926, 1542–1545; *96*, 1927, 404–406; 1126–1128; *97*, 1927, 109–111; Sabin, Brit. Jour. Exp. Path., *15*, 1934, 372–380; Schultz and Hoyt, Jour. Immunol., *15*, 1928, 411–419; Shaffer and Enders, *ibid.*, *37*, 1939, 383–411; Simon, International Clinics, Series 37, *3*, 1927, 123–128; Smith et al., Am. Jour. Path., *17*, 1941, 55–68; Warren et al., Jour. Exp. Med., *71*, 1940, 155–168; Weyer, Proc. Soc. Exp. Biol. and Med., *30*, 1932, 309–313; Zinsser, Jour. Exp. Med., *49*, 1929, 661–670; Zinsser and Seastone, Jour. Immunol., *18*, 1930, 1–9; Zurukzoglu and Hruszek, Cent. f. Bakt. I Abt., Orig., *128*, 1933, 1–12.

2. **Scelus suillum** *spec. nov.* From Latin *suillus*, pertaining to swine.

Common names: Pseudorabies virus, mad-itch virus.

Hosts: Domestic cattle, swine, dog, cat, horse, sheep. Experimentally, also rabbit, guinea pig, white rat, white mouse, gray field mouse, duck, chicken, chick embryo; *Macaca mulatta* (Zimmermann), rhesus monkey.

Geographical distribution: France, Germany, Hungary, Holland, Denmark, Switzerland, Siberia, Brazil, United States.

Induced disease: In cattle, licking of affected area, usually somewhere on hindquarters, sudden decrease in milk production in dairy animals, violent rubbing, biting, and gnawing of lesion; swelling and discoloration of affected parts with oozing of serosanguineous fluid; grinding of teeth and excessive salivation in some individuals; death, preceded by clonic convulsions, violent tossing of head, and shallow respiration, usually 36 to 48 hours after onset. In pig, mild but highly contagious disease; slight nerve-cell degeneration, predominance of vascular and interstitial lesions.

Transmission: By contact in swine, not in cattle. By feeding in cats, brown rats, and swine.

Serological relationships: Cross neutralization between constituent strains. Anti-herpes sera protect in some cases against small, but constantly infective, doses of pseudorabies virus.

Literature: Aujeszky, Cent. f. Bakt. I Abt., Orig., *58*, 1902, 353–357; F. B. Bang, Jour. Exp. Med., *76*, 1942, 263–270;

O. Bang, Acta path. et microbiol. Scand., Suppl., *11*, 1932, 180–182; Carini and Maciel, Bull. Soc. Path. exot., *5*, 1912, 576–578; Følger, Acta path. et microbiol. Scand., Suppl., *11*, 1932, 182–187; Glover, Brit. Jour. Exp. Path., *20*, 1939, 150–158; Gowen and Schott, Am. Jour. Hyg., *18*, 1933, 674–687; Hurst, Jour. Exp. Med., *58*, 1933, 415–433; *59*, 1934, 729–749; *63*, 1936, 449–463; Köves and Hirt, Arch. wissensch. u. prakt. Tierheilk., *68*, 1934, 1–23; Morrill and Graham, Am. Jour. Vet. Res., *2*, 1941, 35–40; Brit. Jour. Exp. Path., *15*, 1934, 372–380; Shope, Proc. Soc. Exp. Biol. and Med., *30*, 1932, 308–309; Jour. Exp. Med., *54*, 1931, 233–248; *57*, 1933, 925–931; *62*, 1935, 85–99, 101–117, Traub, *ibid.*, *58*, 1933, 663–681; *61*, 1935, 833–838.

3. **Scelus beta** *spec. nov.* From *beta*, second letter of Greek alphabet, in reference to common name.

Common name: B virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. *CERCOPITHECIDAE—Macaca mulatta* (Zimmermann), rhesus monkey. Experimentally, also *LEPORIDAE—Oryctolagus cuniculus* (L.), rabbit. *CAVIIDAE—Cavia porcellus* (L.), guinea pig.

Geographical distribution: United States (from captive monkeys and man).

Induced disease: In man, local and relatively insignificant lesion on bitten part, later flaccid paralysis of legs, urinary retention, ascending paralysis, and death by respiratory failure. In *Macaca mulatta*, experimentally by intracutaneous injection, hemorrhagic or vesiculo-pustular lesions without later involvement of central nervous system but with subsequent acquired immunity. Acidophilic intranuclear inclusions in lesions.

Transmission: To man, by bite of monkey. To monkey, experimentally, by injection.

Literature: Burnet et al., Austral. Jour. Exp. Biol. and Med. Sci., *17*, 1939, 35–40, 41–51; Sabin, Brit. Jour. Exp. Path., *15*, 1934, 248–268, 268–279, 321–334; Sabin and Hurst, *ibid.*, *16*, 1935, 133–148; Sabin and Wright, Jour. Exp. Med., *59*, 1934, 115–136.

4. **Scelus tertium** *spec. nov.* From Latin *tertius*, third.

Common name: Virus III of rabbits.

Host: *LEPORIDAE—Oryctolagus cuniculus* (L.), domestic rabbit.

Insusceptible species: No obvious disease in inoculated guinea pig, white mouse, monkey (*Macaca mulatta* Zimmermann), rat, or man; hence the assumption that these are naturally immune, but they may be merely tolerant or klendusic.

Geographical distribution: United States (apparently spontaneous in some individuals of the laboratory rabbit).

Induced disease: In domestic rabbit, experimentally, after incubation period of 4 to 6 days, failure to eat, loss of weight, occasionally diarrhea and temperatures of 104 to 107° F; small, superficial, red spots and papules on skin at site of inoculation; local infiltration of tissues with endothelial leucocytes, swelling of involved epithelial cells; nuclear inclusions present in endothelial leucocytes and some other cells; disease not fatal; virus in circulating blood only during early stages; recovery in a few days without scar formation but with development of specific immunity. The course of the natural disease, presumed to occur in rabbits, is still unknown.

Transmission: Experimentally, by injection of filtrates from diseased tissues; on several occasions also from blood or tissues of apparently normal rabbits.

Serological relationships: Specific neutralizing substances occur in the serum of recovered rabbits.

Immunological relationships: Specific immunity but no cross reactions with vaccinia or herpes viruses.

Thermal inactivation: In 10 minutes at 55° C, but not in 30 minutes at 45° C.

Filterability: Passes Berkefeld V and N filters; passes L_3 filter candle.

Other properties: Viable at least 6

weeks in 50 per cent glycerine and 16 months dried when frozen, and stored on ice.

Literature: Andrewes, Brit. Jour. Exp. Path., *10*, 1929, 188–190, 273–280; Jour. Path. and Bact., *33*, 1930, 301–312; *50*, 1940, 227–234; Rivers and Stewart, Jour. Exp. Med., *48*, 1928, 603–613; Rivers and Tillett, *ibid.*, *39*, 1924, 777–802; *40*, 1924, 281–287; Topacio and Hyde, Am. Jour. Hyg., *15*, 1932, 99–124.

5. **Scelus ulceris** *spec. nov.* From Latin *ulcus*, sore spot.

Common name: Ovine balano-posthitis virus.

Host: *BOVIDAE—Ovis aries* L., sheep.

Geographical distribution: United States, Australia.

Induced disease: In sheep, ulceration with scab production: lesions most severe on prepuce and vulva; in the male, the penis may be involved, usually only with mild inflammation, but if accompanied by paraphimosis there may be extensive ulceration and heavy scab formation.

Transmission: Venereally. Experimentally, by inoculation of prepuce.

Filterability: Passes Berkefeld N and W filters, a 7 lb Mandler candle, and a 3½ per cent collodion membrane.

Literature: Tunnicliff and Matisheck, Science, *94*, 1941, 283–284.

6. **Scelus marmorans** *spec. nov.* From Latin *marmorare*, to marble, in reference to mottling of spleen and liver in host.

Common name: Ectromelia virus.

Hosts: *MURIDAE—Mus musculus* L., white mouse. Esperimentally, also *MURIDAE—Rattus norvegicus* (Berkenhout), rat (infection inapparent). Also, *PHASIANIDAE—Gallus gallus* (L.), chick embryo (12-day-old White Leghorn chick embryo at 36 to 37° C; less satisfactory results at higher temperatures of incubation or in embryos in spring eggs). Derived strains of this virus infect rabbit and guinea pig, not susceptible to original virus from mouse.

Geographical distribution: England.

Induced disease: In white mouse, spleen mottled, liver edges translucent, peritoneal fluid increased in amount; loss of weight; later, cutaneous lesions on foot or elsewhere; affected foot swells, becomes moist, scabbed, then recovers or dries up and separates from the skin at limit of original swelling; in acute disease, death without gross lesions, or, at autopsy, gut dark red, liver dirty gray, soft, bloodless, sometimes mottled, spleen necrotic; inclusion bodies most numerous in lesions of the skin, round or oval, 4 to 13 microns long, without internal differentiation; very young mice probably become infected without developing apparent disease and remain carriers for some time. In rat, inapparent infection; after initial increase of virus, circulating antibodies appear and immunity to reinfection is established.

Transmission: In mouse, by contact. In rat, experimentally, by intranasal inoculation.

Serological relationships: Neutralizing antibodies occur in convalescent mouse serum. Immune sera from the guinea pig specifically agglutinate elementary bodies obtained from infected skin of the white mouse.

Immunological relationships: Recovered mice are solidly immune to many lethal doses.

Thermal inactivation: At 55° C in 30, not in 10, minutes.

Filterability: In broth, passes Mandler, Pasteur-Chamberland L_2, and Berkefeld N filters.

Other properties: Survives drying 6 months, freezing (−10° C) 2 months, 50 per cent glycerine 5 months at least. Resists 1 per cent phenol 20, not 40, days. Size, estimated by filtration, 100 to 150 millimicrons; by ultraviolet-light photography, 130 to 140 millimicrons.

Literature: Barnard and Elford, Proc. Roy. Soc. London, Ser. B, *109*, 1931, 360–380; Baumgartner, Cent. f. Bakt., I Abt., Orig., *133*, 1935, 282–289; Burnet and Lush, Jour. Path. and Bact., *42*, 1936, 469–476; *43*, 1936, 105–120; Jahn, Arch. f. Virusforsch., *1*, 1939, 91–103; Kikuth and

Gönnert, Arch. f. Virusforsch., *1*, 1940, 295–312; Marchal, Jour. Path. and Bact., *33*, 1930, 713–728; McGaughey and Whitehead, *ibid.*, *37*, 1933, 253–256; Paschen, Cent. f. Bakt., I Abt., Orig., *135*, 1936, 445–452.

7. Scelus bovinum *spec. nov.* From Latin *bovinus*, of ox, bull, or cow.

Common name: Erosive-stomatitis virus.

Host: *BOVIDAE—Bos taurus* L., domestic cattle. Experimentally, also chorioallantoic membrane of developing hen's egg.

Insusceptible species: *CAVIIDAE—Cavia porcellus* (L.), guinea pig. (In rats, rabbits, mice, sheep, no reaction has been noted after inoculation.)

Geographical distribution: South Africa (Natal); perhaps Ireland (Armagh-disease virus).

Induced disease: In young domestic cattle, lesions on tongue, dental pad, and lips pearl-like at first, then breaking down to form superficial erosions, with white glistening base and red border. Lesions may coalesce to form large, ragged, eroded areas, healing uneventfully with scar formation. No foot lesions; no excessive salivation; no "hotness" of mouth; no systemic disturbances.

Transmission: Spreads slowly, mainly to animals less than three years old, probably by contact. Experimentally, by injection into dental pads, lips, or tongue. Filterability: Passes Gradocol membrane of about 400 millimicron average pore diameter.

Other properties: Viable after at least 11 days at room temperature, 21 days at refrigerator temperature, 6 weeks frozen and dried in horse-serum saline.

Literature: Mason and Neitz, Onderstepoort Jour. Vet. Sci. and Anim. Indust., *15*, 1940, 159–173.

Genus IV. Hostis *gen. nov.*

Viruses of the Foot-and-Mouth Disease Group, inducing diseases mainly characterized by vesicular lesions. Generic name from Latin *hostis*, enemy or stranger.

The type species is *Hostis pecoris spec. nov.*

Key to the species of genus **Hostis.**

I. Infecting cattle and other animals with cloven hoofs; horse immune or highly resistant. ..... 1. *Hostis pecoris.*

II. Infecting horse readily. ..... 2. *Hostis equinus.*

1. Hostis pecoris *spec. nov.* From Latin *pecus*, cattle.

Common names: Foot-and-mouth disease virus; Virus der Maul- und Klauenseuche.

Hosts: Cow, pig, sheep, goat, reindeer, bison. Experimentally, also guinea pig, rabbit, rat.

Insusceptible species: Chick embryo (chorioallantois); horse (immune or very resistant).

Induced disease: In cow, after incubation period of 2 to 4 days or more, fever, vesicular lesions on tongue, lips, gums, hard palate and feet, soon rupturing; salivation, lameness, generally recovery.

Transmission: Spread rapid, source of infection often obscure; saliva is infective before lesions become obvious.

Thermal inactivation: At 70° C, not at 60° C, in 15 minutes.

Filterability: Passes Seitz, Berkefeld V and N, and Chamberland L_1 filters.

Strains: Three strains, A, O and C, are immunologically distinct from each other.

Other properties: Particle calculated to be about 20 millimicrons in diameter by centrifugation data, 8 to 12 millimicrons in diameter by filtration; may be separated from mixtures with the larger equine vesicular stomatitis virus by differential filtration. Viable after drying

in vacuo, at least a week at −4 to 0° C. Readily destroyed by 1 to 2 per cent sodium hydrate or above pH 11. Soon inactivated near pH 6.0, but moderately stable at pH 2.0 to 3.0; optimum condition for storage at pH 7.5 to 7.7 in absence of air; return from 3.0 to 7.5 inactivates, however.

Literature: Elford and Galloway, Brit. Jour. Exp. Path., *18*, 1937, 155–161; Galloway and Elford, *ibid.*, *14*, 1933, 400–408; *16*, 1935, 588–613; *17*, 1936, 187–204; Galloway and Schlesinger, Jour. Hyg., *37*, 1937, 463–470; Hare, Jour. Path. and Bact., *35*, 1932, 291–293; Loeffler and Frosch, Cent. f. Bakt., I Abt., *23*, 1898, 371–391; Matte and Sanz, Bull. Soc. Path. Exot., *14*, 1921, 523–529; Olitsky and Boez, Jour. Exp. Med., *45*, 1927, 673–683, 685–699, 815–831, 833–848; Pyl, Ztschr. f. physiol. Chemie, *226*, 1934, 18–28; Pyl and Klenk, Cent. f. Bakt., I Abt., Orig., *128*, 1933, 161–171; Schlesinger and Galloway, Jour. Hyg., *37*, 1937, 445–462.

2. **Hostis equinus** *spec. nov.* From Latin *equinus*, pertaining to horses.

Common names: Vesicular-stomatitis virus, equine vesicular stomatitis virus.

Hosts: Horse, domestic cattle. Experimentally, also guinea pig, swine, white mouse, rabbit (relatively resistant), chick embryo; *Macaca mulatta* (Zimmermann), rhesus monkey; *M. irus*, cynomolgus monkey.

Insusceptible species: Chicken (except embryo).

Geographical distribution: United States (Indiana, New Jersey).

Induced disease: In horse, resembles foot-and-mouth disease of cattle; reddened patches on buccal mucosa, moderate fever, salivation, followed by appearance of vesicles, especially on tongue, filled with clear or yellowish fluid; vesicles often coalesce and soon rupture leaving an eroded surface which heals soon in the absence of complications. Experimentally, in chorioallantois of developing chick embryo, primary lesions involve moderate ectodermal proliferation, degeneration, necrosis; mesodermal inflammation; slight endodermal proliferation.

Serological relationships: Strains isolated in different localities give antisera capable of neutralizing heterologous isolates of virus, but homologous antisera neutralize in higher dilutions than do heterologous antisera.

Immunological relationships: No cross immunity with respect to equine encephalomyelitis virus.

Filterability: Passes Seitz filter.

Other properties: May be separated from mixtures with foot-and-mouth disease virus by propagation on chorioallantoic membrane of chick embryo, which will not support increase of the latter virus. Inactivated by 1:50,000 methylene blue in 2 mm layer 13 cm from 300 candle-power lamp in 60 minutes but not in 20 minutes. Particle estimated on the basis of filtration data to be 70 to 100 millimicrons in diameter; 60 millimicrons in diameter by centrifugation. Not destroyed by acidifying to pH 3 and returning to pH 7.5 (difference from foot-and-mouth disease virus).

Literature: Burnet and Galloway, Brit. Jour. Exp. Path., *15*, 1934, 105–113; Cox and Olitsky, Proc. Soc. Exp. Biol. and Med., *30*, 1933, 653–654; Cox et al., *ibid.*, *30*, 1933, 896–898; Elford and Galloway, Brit. Jour. Exp. Path., *18*, 1937, 155–161; Galloway and Elford, *ibid.*, *14*, 1933, 400–408; *16*, 1935, 588–613; Mohler, Jour. Am. Vet. Med. Assoc., *52*, 1918, 410–422; Olitsky et al., Jour. Exp. Med., *59*, 1934, 159–171; Pyl, Ztschr. f. physiol. Chemie, *226*, 1934, 18–28; Sabin and Olitsky, *ibid.*, *66*, 1937, 15–34, 35–57; *67*, 1938, 201–228, 229–249; Syverton et al., Science, *78*, 1933, 216–217.

Genus V. **Molitor** *gen. nov.*

Viruses of the Wart-Disease Group, inducing diseases mainly characterized by tissue proliferation without vesicle or pustule formation. Generic name from Latin *molitor*, contriver.

The type species is *Molitor verrucae spec. nov.*

Key to the species of genus **Molitor.**

I. Affecting man.
1. *Molitor verrucae.*
2. *Molitor hominis.*

II. Affecting cow.
3. *Molitor bovis.*

III. Affecting dog.
4. *Molitor buccalis.*

IV. Affecting chicken.
5. *Molitor tumoris.*

V. Affecting rabbit.
6. *Molitor gingivalis.*
7. *Molitor sylvilagi.*
8. *Molitor myxomae.*

1. **Molitor verrucae** *spec. nov.* From Latin *verruca*, wart.

Common name: Common-wart virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. Perhaps also *BOVIDAE—Bos taurus* L., cow. *CANIDAE—Canis familiaris* L., dog.

Induced disease: Experimentally in man, incubation period long, 4 weeks to 6 or more months; initially acanthosis (overgrowth of prickle cell layer of epidermis) and flattening of the papillae; later, interpapillary hypertrophy, inflammation, and marked hyperkeratosis.

Transmission: By contact; in some cases, venereally. Experimentally, by skin scar fication.

Filterability: Passes Berkefeld N filter.

Literature: Ciuffo, Giorn. ital. d. malattie veneree e d. pelle, *48*, 1907, 12–17; Kingery, Jour. Am. Med. Assoc., *76*, 1921, 440–442; Payne, Brit. Jour. Dermat., *3*, 1891, 185–188; Schultz, Deutsch. med. Wchnschr., *34*, 1908, 423; Serra, Giorn. ital. d. malattie veneree e d. pelle, *65*, 1924, 1808–1814; Ullmann, Acta otolaryngologica, *5*, 1923, 317–334; Wile and Kingery, Jour. Am. Med. Assoc., *73*, 1919, 970–973.

2. **Molitor hominis** *comb. nov.* (***Strongyloplasma hominis*** Lipschütz, Arch. Dermat. u. Syph., *107*, 1911, 395.) From Latin *homo*, man.

Common name: *Molluscum contagiosum* virus.

Host: *HOMINIDAE—Homo sapiens* L., man.

Geographical distribution: Perhaps essentially world-wide.

Induced disease: In man, experimentally, prodromal period may be 14 to 50 days, lesions at first like pimples, becoming red, painful, swollen, developing into small tumors covered with stretched and shiny skin; lesions commonest on face, arms, buttocks, back, and sides, healing spontaneously. Inclusions within epithelial cells, known as molluscum bodies, measure 9 to 24 microns in diameter when approximately spherical, 24 to 27 microns in width and 30 to 37 microns in length when elongated; they contain elementary bodies about 0.3 micron in diameter. The outer envelope of the molluscum body is of carbohydrate.

Transmission: By contact. By fomites.

Filterability: Passes Chamberland L_1 and Berkefeld V filters.

Literature: Goodpasture and King, Am. Jour. Path., *3*, 1927, 385–394; Goodpasture and Woodruff, *ibid.*, *7*, 1931, 1–8; Juliusberg, Deutsch. med. Wchnschr.,

51, 1905, 1598–1599; Lipschütz, Arch. Dermat. u. Syph., *107*, 1911, 387–396; in Kolle, Kraus and Uhlenhuth, Handbuch der Pathogenen Mikroorganismen, *8*, 1930, 1031–1040; Van Rooyen, Jour. Path. and Bact., *46*, 1938, 425–436; *49*, 1939, 345–349; Wile and Kingery, Jour. Cutan. Dis., 37, 1919, 431–446.

3. **Molitor bovis** *spec. nov.* From Latin *bos*, cow.

Common name: Cattle-wart virus.

Host: *BOVIDAE—Bos taurus* L., domestic cattle.

Geographical distribution: United States.

Induced disease: In cattle, especially about head, neck, and shoulders in young animals, on udders in cows, affected skin thickened at first, then rough, nodular; warts sometimes become large and pendulous, adversely affecting growth of host; they sometimes become cauliflower-like tumors several inches in diameter; spontaneous regression is not infrequent. Hides from affected animals are reduced in value.

Transmission: Believed to be through injuries to skin when the injured part comes in contact with warty animals or with rubbing posts, chutes, fences, buildings, or other structures with which affected animals have come in contact previously. Experimentally, by skin inoculations, especially in animals under 1 year of age.

Filterability: Passes Berkefeld N filter.

Literature: Creech, Jour. Agr. Res., *39*, 1929, 723–737; U. S. Dept. Agr., Leaflet 75, 1931, 1–4.

4. **Molitor buccalis** *spec. nov.* From Latin *bucca*, cheek.

Common name: Canine oral-papillomatosis virus.

Host: *CANIDAE—Canis familiaris* L., dog.

Insusceptible species: Cat, rabbit, guinea pig, rat, mouse; *Macaca mulatta* (Zimmermann), rhesus monkey.

Induced disease: In young dog, experimentally, about 1 month after inoculation of buccal membrane by scarification, pale, smooth elevations, becoming gradually more conspicuous and roughened; finally a mass of closely packed papillae is formed. Regression with subsequent immunity is frequent; no scars are left on regression. Secondary warts often appear in other parts of the mouth 4 to 6 weeks after primary warts have first been observed.

Transmission: Experimentally by skin scarification.

Serological relationships: Not inhibited by antiserum effective against common-wart virus of man.

Thermal inactivation: At some temperature between 45 and 58° C in 1 hour.

Filterability: Passes Berkefeld N filter.

Other properties: Viable after freezing and drying, if stored dry in icebox, at least 63 days; in storage in equal parts of glycerine and 0.9 per cent NaCl solution at least 64 days.

Literature: DeMonbreun and Goodpasture, Am. Jour. Path., *8*, 1932, 43–56; M'Fadyean and Hobday, Jour. Comp. Path. and Therap., *11*, 1898, 341–344; Penberthy, *ibid.*, *11*, 1898, 363–365.

5. **Molitor tumoris** *spec. nov.* From Latin *tumor*, swelling.

Common names: Fowl-sarcoma virus, Rous chicken-sarcoma virus.

Hosts: *PHASIANIDAE—Gallus gallus* (L.), chicken. Experimentally, also pheasant (serial transfer difficult) and duck (by cell transfer only but filtrates from duck infect injected chicken).

Insusceptible species: Turkey, guinea fowl (both immune to filtrates but capable of supporting tumor line if alternated in a series with common fowl hosts); geese.

Induced disease: In hen, originally found in an adult, pure-bred hen of Barred Plymouth Rock variety. Experimentally transmitted, a circumscribed nodule soon becomes evident at site of implantation; later this becomes necrotic or cystic at its center; as growth enlarges, host becomes emaciated, cold, somnolent, and finally dies; discrete metastases are often found in lungs, heart, and liver. Parent cell of sarcoma is claimed to be

the normal histiocyte, but virus in the affected fowl is not confined to the sarcoma, being widespread in the body in spleen, liver, muscle, brain, etc. In the chick embryo, serial passage is feasible on the egg membrane, in which focal lesions involve only ectodermal tissue.

Transmission: By injection of affected fowl cells or filtrates. Certain transmissible tar-induced sarcomas, not infecting by filtrates, nevertheless induce the formation of antibodies capable of neutralizing this virus. An inhibitor of the virus extracted from tumors appears to be a protein, inactivated at 65° C, but not at 55° C, in 30 minutes and destroyed by trypsin in 3 to 5 hours at pH 8. Oleic acid also may act as an inhibitor. No spontaneous transmission in chickens kept together.

Serological relationships: Particles sedimented by centrifugal force 20,000 to 30,000 times gravity are specifically agglutinated by sera of fowls bearing corresponding tumor. At least one antigen in tumors of hen and duck not in healthy birds; this one fixes complement and gives cross reactions with Rous, Mill Hill 2, Fujinami, and RFD2 tumors. Virus injected into goats produces two antibodies but only one if previously heated; the antibody to the heat-stable constituent requires complement to neutralize virus; the only antibody produced in ducks does not require complement to neutralize.

Thermal inactivation: At or below 54° C in 20 minutes.

Filterability: Passes Berkefeld V and no. 5 (medium) filters.

Other properties: Particle size estimated as about 100 millimicrons (but some say 50 or even 15 millimicrons) in diameter by filtration through graded membranes, about 70 millimicrons (molecular weight 140,000,000) by ultracentrifugation. Contains 8.5 to 9.0 per cent nitrogen, 1.5 per cent phosphorus. Protein tests positive. Feulgen reaction for thymonucleic acid absent; 10 to 15 per cent of the protein may be nucleic acid, probably of ribose type. Pentose present. Virus believed to be of globulin nature or attached to globulin particles (Lewis and Mendelsohn, Am. Jour. Hyg., *12*, 1930, 686–689). Viable indefinitely in dried spleen as in dried sarcoma tissues.

Strains: Several strains have been studied in addition to the original Rous sarcoma no. 1 strain; immunological relationships have been shown between the original strain, the des Ligneris sarcoma strain, the Fujinami sarcoma strain, the fibrosarcoma MH1 and endothelioma MH2 strains; other isolates also have shown serological interrelationships.

Literature: Amies, Jour. Path. and Bact., *44*, 1937, 141–166; Amies et al., Am. Jour. Cancer, *35*, 1939, 72–79; Andrewes, Jour. Path. and Bact., *34*, 1931, 91–107; *35*, 1932, 407–413; *37*, 1933, 17–25, 27–44; *43*, 1936, 23–33; Claude, Jour. Exp. Med., *66*, 1937, 59–72; Science, *87*, 1938, 467–468; *90*, 1939, 213–214; Am. Jour. Cancer, *37*, 1939, 59–63; Claude and Rothen, Jour. Exp. Med., *71*, 1940, 619–633; Dmochowski and Knox, Brit. Jour. Exp. Path., *20*, 1939, 466–472; Elford and Andrewes, *ibid.*, *16*, 1935, 61–66; Gye and Purdy, Jour. Path. and Bact., *34*, 1931, 116–117 (Abst.); Haddow, *ibid.*, *37*, 1933, 149–155; Helmer, Jour. Exp. Med., *64*, 1936, 333–338; Keogh, Brit. Jour. Exp. Path., *19*, 1938, 1–9; Ledingham and Gye, Lancet, *228*, 1935 (*1*), 376–377; Lewis and Mendelsohn, Am. Jour. Hyg., *12*, 1930, 686–689; des Ligneris, Am. Jour. Cancer, *16*, 1932, 307–321; McIntosh, Jour. Path. and Bact., *41*, 1935, 215–217; Mellanby, Jour. Path. and Bact., *46*, 1938, 447–460; *47*, 1938, 47–64; Mendelsohn et al., Am. Jour. Hyg., *14*, 1931, 421–425; Purdy, Brit. Jour. Exp. Path., *13*, 1932, 473–479; Rous, Jour. Exp. Med., *13*, 1911, 397–411.

6. **Molitor gingivalis** *spec. nov.* From Latin *gingiva*, gum.

Common name: Rabbit oral-papillomatosis virus.

Hosts: *LEPORIDAE—Oryctolagus cuniculus* (L.), domestic rabbit. Experimentally, also *Lepus americanus*

Erxleben, snowshoe hare; *L. californicus* Gray, jack rabbit; *Sylvilagus* sp., cottontail rabbit.

Geographical distribution: United States.

Induced disease: In rabbit, benign papillomas, having the form of small, discrete, gray-white, sessile or pedunculated nodules, usually multiple, on lower surface of tongue or, less frequently, on gums or floor of mouth.

Transmission: Perhaps by mother to suckling young, with a latent period before onset of disease. Not highly contagious, if contagious at all, in old animals. Experimentally by puncture of tissues in the presence of virus.

Immunological relationships: Specific immunity develops as a result of disease, but no cross immunity with respect to rabbit-papilloma virus, which differs also in failing to act on oral mucosa.

Filterability: Passes Berkefeld V and N filters.

Literature: Parsons and Kidd, Jour. Exp. Med., 77, 1943, 233–250.

7. Molitor sylvilagi *spec. nov.* From New Latin *Sylvilagus*, generic name of cottontail rabbit.

Common names: Rabbit papilloma or papillomatosis virus, rabbit wart virus.

Hosts: *LEPORIDAE—Sylvilagus* sp., cottontail rabbit. Experimentally, also *LEPORIDAE—Oryctolagus cuniculus* (L.), domestic rabbit.

Geographical distribution: United States.

Induced disease: In cottontail rabbit, at first minute elevations along lines of scarification; later solid masses of wrinkled keratinized tissue, 3 to 4 millimeters in thickness; eventually cornified warts, striated perpendicularly at top, fleshy at base, 1 to 1.5 cm in height; regression rare; natural papillomas become malignant occasionally. In domestic rabbit, experimentally, blood antibody remains low but virus is always masked, preventing serial passage; discrete lesions on skin permit quantitative tests; tarring causes localization of virus from blood stream; papillomas give rise to malignant acanthomatous tumors by graded continuous alteration; metastasis frequent; transplantation to new hosts successful in series; antibody specific for the virus is formed continuously in the transplanted growths although virus is not directly demonstrable by subinoculation from them; malignant growths appear more promptly and frequently where epidermis has been tarred long; virus appears specific for epithelium of skin; growths disappear if treated with X-rays, 3600 r at one time or fractionally; 60 per cent are cured with 3000 r, but 2000 r ineffective.

Transmission: Experimentally, by scarification of skin. Abnormal susceptibility to infection is noted in rabbit skin treated with 0.3 per cent methylcholanthrene in benzene or equal parts of turpentine and acetone.

Serological relationships: Specific neutralization, reversible on dilution. Complement fixation specific, with virus particle as antigen; no cross reaction with antisera for vaccinia, herpes, fibroma, or myxoma viruses. Precipitates occur in properly balanced mixtures of virus and specific antiserum; virus and antibody in both free and neutralized states are present in both soluble and insoluble phases of these suspensions.

Immunological relationships: Intraperitoneal injections immunize specifically. Rabbits immunized to fibroma and myxoma viruses are susceptible to rabbit papilloma virus.

Thermal inactivation: At 70° C, not at 65 to 67°C, in 30 minutes; in 0.9 per cent sodium chloride solution at 65 to 66° C, time not stated.

Filterability: Passes Berkefeld V, N, and W filters; particle size calculated as 23 to 35 millimicrons by filtration as compared with 32 to 50 millimicrons by centrifugation and 44.0 millimicrons by measurement of electron micrographs, which show the particle to be approximately spherical in shape.

Other properties: Infectious particle has sedimentation constant $S_{20} = ca.\ 250 \times 10^{-13}$ cm per sec. per dyne; usually there is a secondary boundary at about 375×10^{-13}. Isoelectric point between pH 4.8 and 5.1. Maximum absorption at about 2750 Å. Contains thymus nucleic acid about 6.8 to 8.7 per cent; maximum absorption of nucleic acid at about 2630 Å.

Literature: Beard et al., Jour. Inf. Dis., *65*, 1939, 43–52; *69*, 1941, 173–192; Bryan and Beard, *ibid.*, *65*, 1939, 306–321; Friedewald, Jour. Exp. Med., *75*, 1942, 197–220; Hoyle, Jour. Path. and Bact., *50*, 1940, 169–170; Kidd, Jour. Exp. Med., *68*, 1938, 703–724, 725–759; *70*, 1939, 583–604; *71*, 1940, 469–494; *74*, 1941, 321–344; *75*, 1942, 7–20; Kidd and Rous, *ibid.*, *68*, 1938, 529–562; *71*, 1940, 813–838, Kidd et al., *ibid.*, *64*, 1936, 63–77, 79–96; Rous and Beard, *ibid.*, *60*, 1934, 701–722; *62*, 1935, 523–548; Rous and Kidd, *ibid.*, *67*, 1938, 399–428; *71*, 1940, 787–812; Rous et al., *ibid.*, *64*, 1936, 385–400, 401–424; Schlesinger and Andrewes, Jour. Hyg., *37*, 1937, 521–526; Sharp et al., Proc. Soc. Exp. Biol. and Med., *50*, 1942, 205–207; Shope, Jour. Exp. Med., *58*, 1933, 607–624; *65*, 1937, 219–231; Syverton et al., *ibid.*, *73*, 1941, 243–248; Taylor et al., Jour. Inf. Dis., *71*, 1942, 110–114.

8. **Molitor myxomae** (Aragão) *comb. nov.* (*Chlamidozoon myxomae* Aragão, Brazil-Med., *25*, 1911, 471; name later abandoned by its original author in favor of *Strongyloplasma myxomae* Aragão, Mem. Inst. Oswaldo Cruz. *20*, 1927, 231 and 243. The name *Sanarellia cuniculi* Lipschütz, Wien. klin. Wochenschr., *40*, 1927, 1103, was based on the supposed causative organism, defined as varying in size between the size of chlamydozoa and of large cocci; it is not clear whether the structures observed and named were virus particles or not.) From New Latin *myxoma*, a kind of soft tumor, from nature of induced lesion.

Common names: Myxoma virus, *virus myxomatosum*.

Hosts: *LEPORIDAE—Oryctolagus cuniculus* (L.), domestic rabbit. Experimentally, also *Sylvilagus* sp., cottontail rabbit; jack rabbit (once in many trials); *Lepus brasiliensis* (resistant and rarely infected). Also chick embryo and duck embryo.

Insusceptible species: *Lepus californicus* Gray, black-tailed jack rabbit; *L. americanus* Erxleben, varying hare; *Sylvilagus transitionalis* Bangs, cottontail; horse, sheep, goat, cattle, dog (but one reported infected), guinea pig, rat, mouse, fowl, pigeon, duck, cat, hamster, monkey; man (but some conjunctival pain and swelling).

Geographical distribution: South America (Brazil, Uruguay, Argentina), United States (California).

Induced disease: In domestic rabbit, a disease (*myxomatosis cuniculi*) almost always fatal at ordinary room temperatures but not at 36 to 42° C, lesions fewer and regressing after 6 to 8 days at these higher temperatures in most animals. At ordinary temperatures, nodules (edematous tumors) in skin near eyes, nose, mouth, ears, and genitalia; edema of eyelids; conjunctivitis with purulent discharge if skin around eyes is involved. Later marked dyspnea, stertorous breathing, cyanosis, asphyxia. Animals usually die within 1 to 2 weeks of infection. Virus enters bloodstream and invades nervous system at random through walls of blood vessels. Discharges from nose, eyes, and the serous exudates from affected tissues are infectious; urine and feces are not. There are cytoplasmic inclusions in affected epidermal cells. In chick embryo, experimentally, intense inflammation, eventual impairment of circulation and necrosis locally; growth best if embryo is grown at 33 to 35° C and chilled to 25° C for 12 to 18 hours before or after inoculation, lesions being linear and associated with capillaries in ectoderm; virus infects and is recoverable from embryo and depresses hatch.

Transmission: By contact with diseased rabbits or cages recently occupied

by them. Through air for a few inches. Rarely by feeding. Experimentally, by rubbing conjunctiva with a bit of infected tissue or with a platinum loop contaminated from diseased conjunctiva; has been recovered from flies. By injection. By flea, *Ctenopsylla felis* (*PULICIDAE*), rarely.

Serological relationships: An attack of the disease induces the formation of neutralizing antibodies. Cross neutralization by antisera to myxoma and fibroma strains. Complement is fixed with myxoma virus as test antigen in the presence of antisera to myxoma or fibroma strains. Serum of rabbit inoculated with a soluble antigen, a heat-labile protein with isoelectric point near pH 4.5, agglutinates myxoma elementary bodies. A second soluble antigen, also heat labile, appears distinct, inhibiting its own antibody even after inactivation of its precipitating power by exposure at 56° C.

Immunological relationships: Myxoma-recovered domestic rabbits become immune to reinfection; fibroma-strain-recovered animals, although partially immunized, still support myxoma-strain virus introduced into the testicle. Heat-inactivated virus (60° C for 30 minutes) tends to immunize if given intradermally; there is then an allergic local response, less severe generalized disease, delayed death or recovery. If fibroma virus precedes myxoma virus by 48 to 96 hours, there is marked protection.

Thermal inactivation: At 55° C in 10 minutes; at 50° C in 1 hour. A substance thermostabile for 30 minutes at 60 to 75° C, but not at 90° C, is itself unable to produce myxomatous changes after the heat treatment but may do so in combination with fibroma virus, and transmissible myxoma virus is then reconstituted. Although it is supposed by some that this indicates the transformation of fibroma-strain virus into myxoma-strain virus, the possibility that heat-modified myxoma-strain virus is reactivated has not been eliminated.

Filterability: Passes Berkefeld V and N filters; not Chamberland L_5 or L_7 filters.

Other properties: Inactivated above pH 12.0 and below pH 4.0. Withstands drying. Viable at least 3 months at 8 to 10° C.

Literature: Aragão, Brazil-med., *25*, 1911, 471; Mem. Inst. Oswaldo Cruz, *20*, 1927, 225–235; Berry and Dedrick, Jour. Bact., *31*, 1936, 50–51 (Abst.); Berry and Lichty, *ibid.*, *31*, 1936, 49–50 (Abst.); Berry et al., Second International Congress for Microbiology, Report of Proceedings, London, 1936, 96 (Abst.); Fisk and Kessel, Proc. Soc. Exp. Biol. and Med., *29*, 1931, 9–11; Gardner and Hyde, Jour. Inf. Dis., *71*, 1942, 47–49; Hobbs, Am. Jour. Hyg., *8*, 1928, 800–839; Science, *73*, 1931, 94–95; Hoffstadt and Omundson, Jour. Inf. Dis., *68*, 1941, 207–212; Hoffstadt and Pilcher, Jour. Bact., *35*, 1938, 353–367; *39*, 1940, 40–41; Jour. Inf. Dis., *64*, 1939, 208–216; *65*, 1939, 103–112; Hoffstadt et al., *ibid.*, *68*, 1941, 213–219; K. E. Hyde, Am. Jour. Hyg., *23*, 1936, 278–297; R. R. Hyde, *ibid.*, *30* (B), 1939, 37–46, 47–55; Hyde and Gardner, *ibid.*, *17*, 1933, 446–465; *30*, (B), 1939, 57–63; Kessel et al., Proc. Soc. Exp. Biol. and Med., *28*, 1931, 413–414; Lipschütz, Wien. klin. Wchschr., *40*, 1927, 1101–1103; Lush, Austral. Jour. Exp. Biol. and Med. Sci., *15*, 1937, 131–139; *17*, 1939, 85–88; Martin, Austral. Counc. Sci. and Indust. Res., Bull. 96, 1936, 28 pages; Moses, Mem. Inst. Oswaldo Cruz, *3*, 1911, 46–53; Parker and Thompson, Jour. Exp. Med., *75*, 1942, 567–573; Plotz, Compt. rend. Soc. Biol., Paris, *109*, 1932, 1327–1329; Rivers, Jour. Exp. Med., *51*, 1930, 965–976; Rivers and Ward, *ibid.*, *66*, 1937, 1–14; Rivers et al., *ibid.*, *69*, 1939, 31–48; Sanarelli, Cent. f. Bakt., I Abt., *23*, 1898, 865–873; Shaffer, Am. Jour. Hyg., *34* (B), 1941, 102–120; Shope, Jour. Exp. Med., *56*, 1932, 803–822; Smadel et al., *ibid.*, *72*, 1940, 129–138; Splendore, Cent. f. Bakt., I Abt., Orig., *48*, 1909, 300–301; Stewart, Am. Jour. Cancer, *15* Suppl.,

1931, 2013–2028; Swan, Austral. Jour. Exp. Biol. and Med. Sci., *19*, 1941, 113–115.

Strains and substrains: A strain from cottontail rabbits (*Sylvilagus sp.*), differing from typical myxoma virus, has been studied extensively under the name fibroma virus. This strain in turn is recognized as consisting of variants and has been investigated as typical (OA) and inflammatory (IA) substrains, antigenically alike but the latter tending to generalize in domestic rabbits. Fibroma virus is not lethal in domestic rabbits as the type strain almost always is; it appears to lack some antigenic constituents, inducing the formation of agglutinins that give cross reactions with the type but of neutralizing and complement-fixing antibodies that do not. The fibroma strain does not generally appear in the blood stream, as myxoma virus does, and is not contagious, at least it does not spread spontaneously among domestic rabbits as the myxoma strain does; the manner of its spread in wild rabbits in nature is not known. Its particle size has been calculated as 126 to 141 millimicrons by centrifugation, 125 to 175 millimicrons by filtration. (Ahlström, Jour. Path. and Bact., *46*, 1938, 461–472; Andrewes, Jour. Exp. Med., *63*, 1936, 157–172; Hoffstadt and Pilcher, Jour. Inf. Dis., *68*, 1941, 67–72; Hurst, Brit. Jour. Exp. Path., *18*, 1937, 1–30; Austral. Jour. Exp. Biol. and Med. Sci., *16*, 1938, 53–64, 205–208; Hyde, Am. Jour. Hyg., *24*, 1936, 217–226; Ledingham, Brit. Jour. Exp. Path., *18*, 1937, 436–449; van Rooyen, *ibid.*, *19*, 1938, 156–163; van Rooyen and Rhodes, Cent. f. Bakt., I Abt., Orig., *142*, 1938, 149–153; Schlesinger and Andrewes, Jour. Hyg., *37*, 1937, 521–526; Shope, Jour. Exp. Med., *56*, 1932, 793–822; *63*, 1936, 33–41, 43–57, 173–178.

FAMILY III. ERRONACEAE *FAM. NOV.*

Viruses of the Encephalitis Group, inducing diseases mainly characterized by effects on nerve tissues.

Key to the genera of family **Erronaceae.**

I. Viruses of the Typical Encephalitis Group.
Genus I. *Erro*, p. 1248.
II. Viruses of the Poliomyelitis Group.
Genus II. *Legio*, p. 1257.
III. Viruses of the Rabies Group.
Genus III. *Formido*, p. 1263.

Genus I. Erro *gen. nov.*

Viruses of the Typical Encephalitis Group, inducing diseases mainly characterized by injuries to cells of the brain. Vectors of some known to be ticks; dipterous insects may also transmit. Generic name from Latin *erro*, a vagrant.

The type species is *Erro scoticus spec. nov.*

Key to the species of genus **Erro.**

I. Affecting sheep principally, but also man.
1. *Erro scoticus.*
II. Affecting man principally.
2. *Erro silvestris.*
3. *Erro incognitus.*
4. *Erro japonicus.*
5. *Erro nili.*
6. *Erro scelestus.*
III. Affecting horse principally, but also man.
7. *Erro equinus.*
IV. Affecting horse, cow, sheep.
8. *Erro bornensis.*

1. **Erro scoticus** *spec. nov.* From Latin *Scoticus*, Scottish.

Common name: Louping-ill virus.

Hosts: *BOVIDAE—Ovis aries* L., sheep. *HOMINIDAE—Homo sapiens* L., man. Experimentally, also mouse, rat (subclinical infection), chick embryo (discrete primary lesions on chorioallantoic membrane), *Macacus rhesus*, horse, cow, pig.

Insusceptible species: Guinea pig, rabbit.

Geographical distribution: Scotland, northern England.

Induced disease: In sheep, encephalitis characterized by dullness followed by incoordination of movement, frequently with tremors chiefly of the head; salivation, champing of jaws; prostration, coma, death. In man, encephalitis with prompt and complete recovery accompanied by formation of specific neutralizing antibodies. In mouse, experimentally, diffuse encephalomyelitis with mild meningeal involvement; following intracerebral inoculation, fine rhythmical tremor involving neck, nose, and ears, unsteadiness, muscle spasms, respiratory distress, sometimes clonic and rarely tonic convulsions; hind limb paralysis, dribbling of urine, cessation of spontaneous limb movements, death; in mouse inoculated intraperitoneally, virus usually enters central nervous system by way of the olfactory mucosa and olfactory bulb, occasionally by trauma at points of

damage; in mouse inoculated intranasally, virus enters blood and reaches the olfactory bulb where it multiplies to a high concentration before infecting the remainder of the brain and the rest of the nervous system; tends to disappear from the blood after sickness begins but persists in the brain until death from encephalitis. In chick embryo, after inoculation of chorioallantoic membrane, edema and opacity spreading from site of inoculation on membrane of 10-day embryo; in 12-day eggs, discrete primary lesions, sometimes with secondary lesions surrounding them on the inoculated membrane; embryo dies in about 6 days, after showing jaundice, edema, mottling of the liver with necrosis; virus regularly in blood. In monkey, *Macacus rhesus*, progressive cerebellar ataxia; encephalomyelitis with involvement and massive destruction of Purkinje cells in the cerebellum.

Transmission: By ticks, *Rhipicephalus appendiculatus* and *Ixodes ricinus* (*IXODIDAE*). In *Rhipicephalus appendiculatus*, the larva or nymph becomes infected; only a few individuals retain virus until the adult stage; virus does not pass through the egg. Non-viruliferous ticks do not acquire virus by feeding with infective ticks on immune animals. Experimentally, by intracerebral or intraperitoneal injection in mouse; by intranasal instillation in rat, mouse, and monkey.

Serological relationships: Complement fixation and neutralization tests show cross reactions with Russian spring-summer encephalitis virus, but immune serum against louping-ill virus is only partially effective in neutralizing the spring-summer encephalitis virus.

Immunological relationships: Mice are protected against louping-ill virus by vaccination with non-virulent spring-summer encephalitis virus but protection is less effective than for the homologous virus. No cross immunity with respect to Rift Valley fever virus or poliomyelitis virus in *Macacus rhesus*, but immunity with respect to reinfection by louping-ill virus has been demonstrated.

Thermal inactivation: At 58° C in 10 minutes.

Filterability: Passes Berkefeld V, N, and W filters.

Other properties: Viable in broth filtrates after storage at 4° C and pH 7.6 to 8.5 for 70 days. Particle diameter, calculated from ultrafiltration data, 15 to 20 millimicrons.

Literature: Alexander and Neitz, Vet. Jour., *89*, 1933, 320–323; Onderstepoort Jour. Vet. Sci. and Anim. Industr., *5*, 1935, 15–33; Alston and Gibson, Brit. Jour. Exp. Path., *12*, 1931, 82–88; Burnet, Jour. Path. and Bact., *42*, 1936, 213–225; Brit. Jour. Exp. Path., *17*, 1936, 294–301; Burnet and Lush, Austral. Jour. Exp. Biol. and Med. Sci., *16*, 1938, 233–240; Casals and Webster, Science, *97*, 1943, 246–248; Jour. Exp. Med., *79*, 1944, 45–63; Elford and Galloway, Jour. Path. and Bact., *37*, 1933, 381–392; Findlay, Brit. Jour. Exp. Path., *13*, 1932, 230–236; Fite and Webster, Proc. Soc. Exp. Biol. and Med., *31*, 1934, 695–696; Galloway and Perdrau, Jour. Hyg., *35*, 1935, 339–346; Hurst, Jour. Comp. Path. and Therap., *44*, 1931, 231–245; M'Fadyean, Jour. Comp. Path. and Therap., *7*, 1894, 207–219; *13*, 1900, 145–154; Pool et al., *ibid.*, *43*, 1930, 253–290; Rivers and Schwentker, Jour. Exp. Med., *59*, 1934, 669–685; Schwentker et al., *ibid.*, *57*, 1933, 955–965.

2. **Erro silvestris** *spec. nov.* From Latin *silvestris*, of the forest, in reference to incidence of the induced disease almost exclusively in those who enter forest lands.

Common names: Spring-summer encephalitis virus, forest spring encephalitis virus.

Hosts: Man; probably cattle, horse; *Eutamias asiaticus orientalis*, *Evotomys rufocanus arsenjevi*. Experimentally, also white mouse, *Macacus rhesus*, birds, goat, sheep, *Microtus michnoi pelliceus* Thom., *Cricetulus furunculus*.

Geographical distribution: Union of Soviet Socialist Republics.

Induced disease: In man, acute non-suppurative encephalitis, abrupt onset, steep rise of temperature to 38 to 40° C, severe headache, giddiness, and vomiting; pareses and paralyses of upper or lower limbs or muscles of neck and back; residual atrophic paralyses common; mortality among cases, 30 per cent; 80 per cent of all cases occur in May and June.

Transmission: By tick, *Ixodes persulcatus* (*IXODIDAE*); the virus seems to hibernate in this species and has proved capable of passing through eggs to progeny. Experimentally, also by ticks *Dermacentor silvarum* and *Haemaphysalis concinna* (*IXODIDAE*).

Serological relationships: Virus-neutralizing antibodies, found without other evidence of disease in some men and in many cattle and horses, believed to indicate susceptibility of these hosts to latent infections. No cross neutralization with St. Louis encephalitis virus. Japanese summer encephalitis virus is in part antigenically related, but some antigenic constituents of this virus are missing in spring-summer encephalitis virus and *vice versa*.

Immunological relationships: Formolized virus immunizes specifically.

Filterability: Passes Berkefeld and Chamberland filter candles.

Literature: Smorodintseff, Arch. f. gesamt. Virusforsch., *1*, 1940, 468–480; Soloviev, Acta Med. U. R. S. S., *1*, 1938, 484–492 (Biol. Abst., *17*, 1943, 1726, no. 18777).

3. **Erro incognitus** *spec. nov.* From Latin *incognitus*, unknown, in reference to mystery surrounding the nature and relationships of this virus, as evidenced by common name.

Common name: Australian X-disease virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. Experimentally, also sheep, horse, cow, rhesus monkey.

Geographical distribution: Australia.

Induced disease: In man, polioencephalitis, especially in children, occurring in late summer; mortality high; characterized by headache, body pains, drowsiness, weakness, then vomiting, fever, convulsions; paralysis of limbs, eye-muscles, or face rare; recovery rapid in non-fatal cases.

Literature: Kneebone, Austral. Jour. Exp. Biol. and Med. Sci., *3*, 1926, 119–127; Perdrau, Jour. Path. and Bact., *42*, 1936, 59–65.

4. **Erro japonicus** *spec. nov.* From New Latin *Japonia*, Japan.

Common name: Japanese B encephalitis virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. Experimentally, also young sheep, mouse, and *Macacus rhesus*.

Geographical distribution: Japan, Union of Soviet Socialist Republics.

Induced disease: In man, loss of appetite, drowsiness, nausea, then rapid rise of temperature, pains in joints and chest; restlessness followed by apathy, coma; death, usually before end of second week, or recovery, sometimes with persistence of evidences of damage done to the nervous system by the disease.

Serological relationships: Specific antiserum does not neutralize St. Louis encephalitis virus or louping-ill virus. Russian autumn-encephalitis virus induces the formation of antisera neutralizing Japanese B encephalitis virus. Russian spring-summer encephalitis virus contains some, but not all, antigens in common with this virus. Australian X-disease virus is distinct in neutralization tests.

Immunological relationships: Specific immunity as a result of earlier infection in mice; no cross protection with respect to St. Louis encephalitis virus. Vaccination with Japanese B encephalitis virus does not enhance resistance to West Nile encephalitis virus but only to the homologous virus.

rat. *MUSCICAPIDAE—Turdus migratorius* L., robin. *PHASIANIDAE—Gallus gallus* (L.), chicken; *Lophortyx californica*, California quail. *PICIDAE —Asyndesmus lewis*, Lewis woodpecker; *Colaptes cafer* (Gm.), red-shafted flicker. *STRIGIDAE—Bubo virginianus* (Gm.), great horned owl. *SUIDAE—Sus scrofa* L., pig. Experimentally, white mouse (some substrains of the Swiss white mouse are genetically more readily infected than others); *Macacus rhesus*; pigeon (inapparent infection); chick embryo and to a limited extent the young hatched chick.

Insusceptible species: Laboratory rabbit, *Cebus* monkey, guinea pig, rat.

Geographical distribution: United States.

Induced disease: In man, during summer and fall, about 9 to 21 days after exposure, headache, high fever, rigidity of neck, tremors; encephalitis, usually with fever; some patients become drowsy, others sleepless or delirious; usual sequelae headaches, irritability, some loss of memory, and drowsiness; neutralizing antibodies maintained *in vivo* at least 2½ years after occurrence of disease. Experimentally, in susceptible strains of white mouse inoculated by intracerebral injection, after 3 to 4 days, coarse tremors, convulsions, prostration, death; perivascular accumulations of mononuclear leucocytes throughout brain, stem, cord, and pia, with destruction of pyramidal cells in the *lobus piriformis* and *cornu Ammonis*; subcutaneous and intraperitoneal injections immunize against subsequent infection by intracerebral inoculation, virus reaching only blood and spleen in the process of immunization unless an excessive dose is given; some substrains of the White Swiss mouse are relatively resistant to infection, requiring inoculation with about 1000 times the minimal infective dose for highly susceptible strains and when infected proving relatively poor sources of virus for subinoculation; highly susceptible substrains of the White Swiss mouse lack a single major, dominant, genetic factor that is present in resistant substrains.

Transmission: By mosquito, *Culex tarsalis* Coquillett (*CULICIDAE*), probably extensively; this insect has been collected in nature carrying the virus. Experimentally, by larvae of American dog tick, *Dermacentor variabilis* (Say) (*IXODIDAE*); by mosquito, *Culex pipiens* Linn., var. *pallans* Coq. (*CULICIDAE*). To mice, by feeding on infected tissues.

Serological relationships: Human antisera may neutralize virus after clinical and subclinical attacks.

Immunological relationships: Specific intracerebral immunity after vaccination by subcutaneous or intraperitoneal injection in mice appears early (about 1 week after vaccination) and disappears before humoral antibody titer reaches its maximum.

Thermal inactivation: At 56° C in 30 minutes.

Filterability: Passes Berkefeld V and N filter candles and collodion membranes 66 millimicrons in average pore diameter.

Other properties: Storage in human brain tissue in glycerine inactivates this virus in about 32 days. Diameter of infective particle calculated from filtration data as about 20 to 33 millimicrons. In storage, rabbit and sheep sera act to some extent as preservatives. At 4° C, after drying *in vacuo* while frozen, viable in apparently undiminished titer for at least 17 months.

Literature: Bang and Reeves, Jour. Inf. Dis., *70*, 1942, 273–274; Bauer et al., Proc. Soc. Exp. Biol. and Med., *31*, 1934, 696–699; Blattner and Cooke, Jour. Inf. Dis., *70*, 1942, 226–230; Blattner and Heys, Proc. Soc. Exp. Biol. and Med., *48*, 1941, 707–710; Cook, Jour. Inf. Dis., *63*, 1938, 206–216; Cook and Hudson, *ibid.*, *61*, 1937, 289–292; Elford and Perdrau, Jour. Path. and Bact., *40*, 1935, 143–146; Hammon and Howitt, Am. Jour. Hyg., *35*, 1942, 163–185; Hammon et al., Science, *94*, 1941, 305–307, 328–330; Jour. Inf. Dis., *70*, 1942, 263–266, 267–272, 278–

283; Harford and Bronfenbrenner, Jour. Inf. Dis., *70*, 1942, 62–68; Harrison and Moore, Am. Jour. Path., *18*, 1937, 361–375; Hodes, Jour. Exp. Med., *69*, 1939, 533–543; Hodes and Webster, *ibid.*, *68*, 1938, 263–271; Lennette and Smith, Jour. Inf. Dis., *65*, 1935, 252–254; Mitamura et al., Trans. Soc. Path. Jap., *27*, 1937, 573–580; Muckenfuss *et al.*, U. S. Pub. Health Service, Public Health Rept., 48, 1933, 1341–1343; O'Leary et al., Jour. Exp. Med., *75*, 1942, 233–246; Reeves et al., Proc. Soc. Exp. Biol. and Med., *50*, 1942, 125–128; Sulkin et al., Jour. Inf. Dis., *67*, 1940, 252–257; Webster, Jour. Exp. Med., 65, 1937, 261–286; *68*, 1938, 111–124; Webster and Clow, *ibid.*, *63*, 1936, 433–448, 827–845; Webster and Fite, *ibid.*, *61*, 1935, 103–114, 411–422; Webster and Johnson, *ibid.*, *74*, 1941, 489–494; Webster et al., *61*, 1935, 479–487; *62*, 1935, 827–847.

7. **Erro equinus** *spec. nov.* From Latin *equinus*, pertaining to horses.

Common name: Equine encephalitis virus.

Hosts: *EQUIDAE—Equus caballus* L., horse; F_1 hybrid of the horse and *E. asinus* L., mule. *HOMINIDAE—Homo sapiens* L., man. *COLUMBIDAE—Columba livia*, domestic pigeon. *PHASIANIDAE*—ring-necked pheasant. *TETRAONIDAE—Tympanuchus cupido* L., var. *americanus* (Reichenbach), prairie chicken. Many additional species have been found to show neutralizing antisera at times and these are presumably natural hosts of the virus upon occasion; among them are: *ANATIDAE—Anas platyrhyncha* L., Mallard and Pekin ducks; *Anser anser* (L.), domestic goose. *BOVIDAE—Bos taurus* L., cow; *Capra hircus* L., goat; *Ovis aries* L., sheep. *CANIDAE—Canis familiaris* L., dog. *CHARADRIIDAE—Oxyechus vociferus* L., killdeer. *CRICETIDAE—Microtus montanus* (Peale), field mouse; *Peromyscus maniculatus* (Wagner), white-footed mouse. *FALCONIDAE—Falco sparverius* L., sparrow hawk. *MELEAGRIDAE—Meleagris gallopavo* L., turkey. *MURIDAE—Rattus rattus* L., black rat. *MUSCICAPIDAE—Turdus migratorius* L., robin. *MUSTELIDAE—Mustela frenata* Lichtenstein, weasel. *PHASIANIDAE—Gallus gallus* (L.), chicken; *Lophortyx californica*, California quail; *Phasianus colchicus* L., ring-necked pheasant. *PICIDAE—Colaptes cafer* (Gm.), red-shafted flicker. *STRIGIDAE—Bubo virginianus* (Gm.), great horned owl. *SUIDAE—Sus scrofa* L., pig. Experimentally, also chick embryo, goose embryo, pheasant embryo, robin embryo, pigeon embryo, turkey embryo, sparrow embryo, duck embryo, and guinea-fowl embryo; white mouse, guinea pig, rabbit, pigeon, white rat, calf, sheep, monkey, goat, dog, hen, turkey; *Zonotrichia leucophrys gambeli*, Gambel sparrow; *Passer domesticus* L., English sparrow; *Lophortyx californica*, quail; *Junco oreganus*, junco; *Toxostoma lecontei lecontei*, thrasher; *Citellus richardsonii* (Sabine), gopher or Richardson's ground squirrel; *Sigmodon hispidus* Say and Ord, cotton rat; *Dipodomys heermanni* Le Conte, kangaroo rat; *Reithrodontomys megalatus*, wild mouse; *Microtus montanus*, *M. californicus* and *M. mordax*, wild mice; *Peromyscus maniculatus* (Wagner), white-footed mouse; *Neotoma fuscipes* Baird, wood rat; *Sylvilagus bachmani* (Waterhouse), brush rabbit; *S. audubonii* (Baird), cottontail rabbit; *Canis familiaris* L., dog (puppies); *Anser cinereus*, goose; *Anas boscas* L., duck; *Circus rufus* (Gm.), hawk; *Turdus merula* L., blackbird; *Ciconia ciconia* L., white stork; *Vultur fulvus* Briss., tawny vulture; *Marmota monax* (L.), woodchuck; *Microtus pennsylvanicus* (Ord.), field vole; *Speotyto cunicularia hypugaea* (Bonaparte), western burrowing owl; *Molothrus ater* (Boddaert), cowbird; common quail or bob-white.

Insusceptible species: Frog (cat and opossum reported as "refractory").

Geographical distribution: United States, Canada, Argentina.

Induced disease: In horse, initial fever, then signs of fatigue, somnolence; occa-

sional excitability followed by incoordinated action of limbs, disturbed equilibrium, grinding of teeth, paresis and varied paralyses; frequently inability to swallow, paralysis of lips and bladder, amaurosis; case fatality about 50 per cent; recovery without sequelae in mild cases; death within 3 to 8 days in severe cases. In man (children particularly vulnerable), a profound, acute, disseminate and focal encephalomyelitis characterized by intense vascular engorgement, perivascular and parenchymatous cellular infiltration and extreme degenerative changes in the nerve cells. In chick embryo, excessive increase of virus continuing until just before host's death, virus being found eventually throughout the egg but most concentrated in the embryo; vaccines made from virus grown in chick embryo and then inactivated are especially effective because of the high titer of virus represented in them; increased resistance with age characteristic of chorioallantoic membrane as well as of hatched chick; rounded acidophilic masses occur usually near periphery of nucleus in embryonic nerve cells; no such inclusions are found as a result of infection with Borna disease virus or poliomyelitis virus.

Transmission: Experimentally by tick, *Dermacentor andersoni* Stiles (*IXODIDAE*), passing through eggs to offspring; this tick is infective to susceptible animals on which it feeds as larva, nymph or adult. Experimentally by *Aēdes aegypti* L. (to guinea pig and horse, preinfective period 4 to 5 days; insects retain virus for duration of life; not to eggs of infected mosquitoes; not passed from males to females or by males from female to female), *A. albopictus*, *A. atropalpus*, *A. cantator*, *A. dorsalis*, *A. nigromaculis*, *A. sollicitans*, *A. taeniorhynchus*, *A. triseriatus*, and *A. vexans* (*CULICIDAE*). *Triatoma sanguisuga* (Le Conte) (*REDUVIIDAE*) has been found infected in nature and has transmitted virus experimentally to guinea pigs. The American dog tick, *Dermacentor variabilis* Say (*IXODIDAE*) has been infected by inoculation, not by feeding; it has not been shown to transmit.

Serological relationships: Neutralizing antibodies are formed as a result of vaccination with inactive, formolized virus; antigenicity of formalin-inactivated virus as well as of active virus is blocked in the presence of antiserum. In rabbit, cerebral resistance is coincident with presence of neutralizing antibody in spinal fluid. In guinea pig, therapy with specific antiserum ineffective if begun after onset of encephalitis; effective if begun within 24 to 48 hours of peripheral inoculation. No cross neutralization reaction with lymphocytic choriomeningitis virus, Japanese B encephalitis virus or St. Louis encephalitis virus. Constituent strains (typical Western and Eastern) do not give cross neutralization reactions, but do show the presence of common antigens by cross reactions in complement fixation not shared with such other viruses as Japanese B encephalitis virus, St. Louis encephalitis virus, West Nile encephalitis virus, lymphocytic choriomeningitis virus. Sera of human cases may be negative by complement fixation tests a few days after onset, yet strongly strain-specific during second week of illness.

Immunological relationships: Young of immunized guinea pigs are immune to homologous strain at least a month after birth. No cross immunity between Western and Eastern strains of equine encephalitis virus.

Thermal inactivation: At 60° C, not at 56° C, in 10 minutes.

Filterability: Passes collodion membranes 66, not 60, millimicrons in average pore diameter. Passes Berkefeld V, N, and W, finest Mandler, and Seitz filters.

Other properties: Inactivated below pH 5.5. Viable at least a year, dry in vacuum. Particle diameter estimated from filtration experiments to be 20 to 30 millimicrons. Electron micrographs show particles as spherical or disk-shaped, about 39 millimicrons in diameter with round or oval region of high density within each; older preparations show

comma-shaped particles. Sedimentation constant, mean $265.5 \times 10^{-13} \pm 5.4 \times 10^{-13}$ (range 252 to 276×10^{-13}). Specific volume 0.864. Molecular weight of lipo-nucleoprotein complex behaving as the virus calculated as 152 million, approximately 250 particles giving 50 per cent infection; material contains 4 per cent carbohydrate. Absorption of ultraviolet light reaches a peak at about 2600 Å., a broad minimum at about 2450 Å., and an increase at 2200 Å.

Strains: The Western strain (so-called Western equine encephalitis virus) may be considered as type of a large group of variants met in nature; some produce clinically milder disease than others (Birch, Am. Jour. Vet. Res., *2*, 1941, 221–226); they may change in virulence on passage in experimental hosts. The Eastern strain (so-called Eastern equine encephalitis virus) has been studied extensively also, and has been found to differ from the type strain especially: in more rapid course of induced disease in the horse; in being experimentally transmissible to sheep, pig, dog, cat and the European hedgehog; in its localization in eastern coast states and absence from the area between California and Wisconsin, where the type strain is found; in failure experimentally to infect *Aëdes aegypti* unless inoculated into body cavity by needle puncture, whereupon it persists and can be transmitted; and in failure of cross-neutralization with the western strain. A strain produced by serial passage in pigeons is reported to have caused no obvious reaction in horses but to have induced the formation of neutralizing antibodies. A Venezuelan strain differs from the type in complement-fixation reactions; it induces in man a mild disease, characterized by malaise, fever, headache or drowsiness, and uneventful recovery (Casals et al., Jour. Exp. Med., 77, 1943, 521–530).

Literature: Bang, Jour. Exp. Med., *77*, 1943, 337–344; Bauer et al., Proc. Soc. Exp. Biol. and Med., *33*, 1935, 378–382; Beard et al., Science, *87*, 1938, 490; Birch, Am. Jour. Vet. Res., *2*, 1941, 221–226; Casals and Palacios, Science, *94*, 1941, 330; Covell, Proc. Soc. Exp. Biol. and Med., *32*, 1934, 51–53; Cox, *ibid.*, *33*, 1936, 607–609; Cox and Olitsky, Jour. Exp. Med., *63*, 1936, 745–765; *64*, 1936, 217–222, 223–232; Cox et al., U. S. Pub. Health Service, Public Health Rept., *56*, 1941, 1905–1906; Davis, Am. Jour. Hyg., *32* (C), 1940, 45–59; Eklund and Blumstein, Jour. Am. Med. Assoc., *111*, 1938, 1734–1735; Feemster, Am. Jour. Public Health, *28*, 1938, 1403–1410; Finkelstein et al., Jour. Inf. Dis., *66*, 1940, 117–126; Fothergill and Dingle, Science, *88*, 1938, 549–550; Fothergill et al., New England Jour. Med., *219*, 1938, 411; Giltner and Shahan, Science, *78*, 1933, 63–64; Jour. Am. Vet. Med. Assoc., *88*, (N.S. *41*), 1936, 363–374; Graham and Levine, Am. Jour. Vet. Res., *2*, 1941, 430–435; Grundmann et al., Jour. Inf. Dis., *72*, 1943, 163–171; Havens et al., Jour. Exp. Med., *77*, 1943, 139–153; Higbie and Howitt, Jour. Bact., *29*, 1935, 399–406; Howitt, Jour. Inf. Dis., *55*, 1934, 138–149; *61*, 1937, 88–95; *67*, 1940, 177–187; Science, *88*, 1938, 455–456; Howitt and Van Herick, Jour. Inf. Dis., *71*, 1942, 179–191; Kelser, Jour. Am. Vet. Med. Assoc., *82*, 1933, 767–771; King, Jour. Exp. Med., *71*, 1940, 107–112; *76*, 1942, 325–334; Kitselman and Grundmann, Kansas Agr. Exp. Sta., Tech. Bull. 50, 1940, 1–15; Merrill and TenBroeck, Jour. Exp. Med., *62*, 1935, 687–695; Meyer et al., Science, *74*, 1931, 227–228; Mitchell et al., Canadian Jour. Comp. Med., *3*, 1939, 308–309; Morgan, Jour. Exp. Med., *74*, 1941, 115–132; Morgan et al., *ibid.*, *76*, 1942, 357–369; Olitsky et al., *ibid.*, *77*, 1943, 359–374; Remlinger and Bailly, Compt. rend. Soc. Biol., Paris, *121*, 1936, 146–149; *122*, 1936, 518–519; *123*, 1936, 562–563; Sabin and Olitsky, Proc. Soc. Exp. Biol. and Med., *38*, 1938, 597–599; Sellards et al., Am. Jour. Hyg., *33* (B), 1941, 63–68; Shahan and Eichhorn, Am. Jour. Vet. Res., *2*, 1941, 218–220; Sharp et al., Proc. Soc. Exp. Biol. and Med., *51*, 1942, 206–207; Arch. Path., *36*, 1943, 167–176; Syverton and Berry, *ibid.*, *34*, 1936, 822–

824; Jour. Bact., *33*, 1937, 60; Am. Jour. Hyg., *32* (B), 1940, 19–23; *33* (B), 1941, 37–41; Jour. Exp. Med., *73*, 1941, 507–529; Taylor et al., Jour. Inf. Dis., *67*, 1940, 59–66; *69*, 1941, 224–231; *72*, 1943, 31–41; TenBroeck, Arch. Path., *25*, 1938, 759 (Abst.); TenBroeck and Merrill, Proc. Soc. Exp. Biol. and Med., *31*, 1933, 217–220; TenBroeck et al., Jour. Exp. Med., *62*, 1935, 677–685; Traub and Ten Broeck, Science, *81*, 1935, 572; Tyzzer and Sellards, Am. Jour. Hyg., *33* (B), 1941, 69–81; Tyzzer et al., Science, *88*, 1938, 505–506; van Roekel and Clarke, Jour. Am. Vet. Med. Assoc., *94* (N.S. *47*), 1939, 466–468; Webster and Wright, Science, *88*, 1938, 305–306; Wesselhoeft et al., Jour. Am. Med. Assoc., *111*, 1938, 1735–1740; Wright, Am. Jour. Hyg., *36*, 1942, 57–67.

8. **Erro bornensis** *spec. nov.* From Borna, name of a town in Saxony where a severe epizootic occurred in 1894 to 1896.

Common name: Borna-disease virus.

Hosts: Horse, cow, sheep, perhaps deer. Experimentally, also rabbit, guinea pig, rat (more susceptible when old than when younger), mouse; *Macaca mulatta* (Zimmermann), rhesus monkey.

Insusceptible species: Ferret, cat, pigeon; probably dog.

Geographical distribution: Würtemburg, Germany, North and South America, Hungary, Russia, Belgium, France, Italy, Roumania.

Induced disease: In horse, encephalomyelitis characterized by lassitude, indifference to external stimuli; later intermittent excitement, difficulty in mastication and deglutition, spasms in various muscles, champing, excessive salivation; pupils unequal in size; paralysis of hindquarters, tail, muscles of tongue, or muscles of back; temperature usually normal; death in 20 to 37 hours or, less often, recovery after about 1 to 3 weeks. Virus may pass placenta and infect fetus in pregnant animals.

Transmission: To rabbit, experimentally by feeding and by injection intracerebrally, intraocularly, nasally, intravenously, subcutaneously, or intraperitoneally; not by living in same cage.

Immunological relationships: No cross immunity conferred by the Western strain of equine encephalomyelitis virus. Isolate of Borna disease virus from the horse immunizes rabbits against isolate from sheep, and *vice versa*. Herpes and rabies viruses do not immunize rabbits against subsequent infection by Borna disease virus.

Thermal inactivation: At 50 to 57° C in 30 minutes; at 70° C in 10 minutes.

Filterability: Passes Berkefeld N and Mandler filters, but with difficulty. Passes collodion membranes of average pore diameter 400 millimicrons readily, 200 millimicrons with difficulty, 175 millimicrons not detectibly. May be separated by differential filtration from louping-ill virus, which will pass even a 125-millimicron membrane.

Other properties: Particle size estimated from filtration data as 85 to 125 millimicrons. Optimum pH for stability in broth at 15 to 20° C is 7.4 to 7.6; very sensitive to greater alkalinity. Viable after 327 days dry at laboratory temperatures. Viable at least 6 months in 50 per cent glycerine. Inactivated by putrefaction in 5 days; by 1 per cent carbolic acid in 4, not in 2, weeks.

Literature: Barnard, Brit. Jour. Exp. Path., *14*, 1933, 205–206; Covell, Proc. Soc. Exp. Biol. and Med., *32*, 1934, 51–53; Elford and Galloway, Brit. Jour. Exp. Path., *14*, 1933, 196–205; Howitt and Meyer, Jour. Infect. Dis., *54*, 1934, 364–367; Nicolau and Galloway, Brit. Jour. Exp. Path., *8*, 1927, 336–341, and in Medical Research Council, Special Report Series No. 121, London, 1928, 90 pp., Ann. Inst. Pasteur, *44*, 1930, 673–696; *45*, 1930, 457–523; Zwick et al., Ztschr. Infektionskr. parasit. Krankh. u. Hyg. d. Haustiere, *30*, 1926, 42–136; *32*, 1927, 150–179.

Genus II. **Legio** *gen. nov.*

Viruses of the Poliomyelitis Group, often recoverable from feces of infected hosts, probably because of involvement of some part of the alimentary tract; usually there is also obvious involvement of some part of the nervous system. Generic name from Latin *legio*, an army or legion.

The type species is *Legio debilitans spec. nov.*

Key to the species of genus **Legio.**

I. Affecting man (see also IV below).
1. *Legio debilitans.*
2. *Legio erebea.*
3. *Legio simulans.*

II. Latent in, or affecting, mouse.
4. *Legio muris.*

III. Affecting birds.
5. *Legio gallinae.*

IV. Affecting swine and swineherds.
6. *Legio suariorum.*

1. **Legio debilitans** *spec. nov.* From Latin *debilitare*, to weaken or maim.

Common names: Poliomyelitis virus, virus of infantile paralysis.

Hosts: *HOMINIDAE—Homo sapiens* L., man. Experimentally, *Cercopithecus aethiops sabaeus*, green African monkey; *Macaca mordax*; *M. mulatta*, the rhesus monkey; *M. irus*, the cynomolgus monkey; mona monkey; for some isolates, *Sigmodon hispidus* Say and Ord, cotton rat; mouse; guinea pig; white rat.

Insusceptible species: Sheep ("refractory" but forms neutralizing antibodies), chicken.

Geographical distribution: Almost world-wide.

Induced disease: In man, probably subclinical in most cases, in view of the presence of specific antibodies in sera from the great majority of adults in all parts of the world; virus probably infects some part of the alimentary tract, being found in stools of most clinical cases, of most apparently healthy contacts, and even of some individuals who have recovered from abortive attacks (in one case 123 days after attack); clinical disease, largely in children, is characterized by invasion of central nervous system, with effects ranging from sore throat, fever, vomiting, and headache to sudden and severe paralysis; the muscles most often involved are those of the legs, but there may be paralysis of abdominal or intercostal muscles. Virus not in urine or saliva, rarely in nasal washings; more often in stools of young than of old patients; in walls of pharynx, ileum, descending colon. Virus has been recovered from sewage. Incidence and fatality affected by racial characteristics, the first lower and the second higher in negroes than in whites in the United States. In monkey, similar disease, no virus in blood, relapse with reappearance of virus reported; in isolated intestinal loops, infection does not occur through normal mucosa in absence of intestinal contents; disease more severe in summer than in autumn, in autumn than in winter; more severe in older than in younger monkeys; no immunity follows inoculation unless obvious disease occurs.

Transmission: Transmission in milk has been suspected and at times confirmed. Virus has been recovered from mixed samples of flies in an epidemic area. No definite arthropod vector has been incriminated. Experimentally, in *Cercopithecus aethiops sabaeus*, the green

African monkey, by intracerebral, intranasal, and intraabdominal inoculation.

Serological relationships: Specific neutralizing antibodies arise after experimental infection in monkeys, but reinfection is not prevented; only a minority of human convalescent sera neutralize virus *in vitro*, the most potent sera probably being obtained from those with transient or light paralysis. Cross neutralization between monkey-passage and murine (cotton-rat and mouse) strains. No cross-neutralization reaction with lymphocytic choriomeningitis virus. Isolates differ somewhat antigenically, homologous titers being higher than heterologous titers in some neutralization tests.

Thermal inactivation: At or below 75° C in 30 minutes.

Filterability: Passes membrane about 35, not 30, millimicrons in average pore diameter.

Other properties: Infectivity of virus maintained well at −76° C or in glycerine but poorly when dried or just frozen. Inactivated readily by hydrogen peroxide. Particle diameter estimated as about 12 millimicrons by filtration studies. Precipitated by half-saturated ammonium sulphate solutions. Electron micrographs show elliptical particles 20 to 30 millimicrons in diameter; impure infectious materials show long threads 20 by 75 to 500 millimicrons in size. Component probably virus has sedimentation constant $S_{20^\circ} = 62 \times 10^{-13}$ cm per sec. per dyne. Inactivated by potassium hydroxide, copper sulfate and potassium permanganate. Stable from pH 2.2 to 10.4 for 2 hours at 37° C.

Literature: Armstrong and Harrison, U. S. Pub. Health Service, Public Health Rept., *50*, 1935, 725–730; Aycock, Am. Jour. Hyg., *7*, 1927, 791–803; Burnet and Jackson, Austral. Jour. Exp. Biol. and Med. Sci., *17*, 1939, 261–270; *18*, 1940, 361–366; Burnet et al., *ibid.*, *17*, 1939, 253–260, 375–391; Elford et al., Jour. Path. and Bact., *40*, 1935, 135–141; Flexner, Jour. Exp. Med., *62*, 1935, 787–804; *63*, 1936, 209–226; *65*, 1937, 497–513; Gard, *ibid.*, *71*, 1940, 779–785; Gordon and Lennette, Jour. Inf. Dis., *64*, 1939, 97–104; Harmon, *ibid.*, *58*, 1936, 331–336; Heaslip, Austral. Jour. Exp. Biol. and Med. Sci., *16*, 1938, 285–286; Howitt, Jour. Inf. Dis., *51*, 1932, 565–573; *53*, 1933, 145–156; Hudson and Lennette, Am. Jour. Hyg., *17*, 1933, 581–586; Jungeblut and Bourdillon, Jour. Am. Med. Assoc., *123*, 1943, 399–402; Jungeblut and Sanders, Jour. Exp. Med., *72*, 1940, 407–436; *76*, 1942, 127–142; Jungeblut et al., *ibid.*, *75*, 1942, 611–629; *76*, 1942, 31–51; Kessel et al., Am. Jour. Hyg., *27*, 1938, 519–529; Jour. Exp. Med., *74*, 1941, 601–609; Kolmer et al., Jour. Inf. Dis., *61*, 1937, 63–68; Kramer et al., Jour. Exp. Med., *69*, 1939, 46–67; Lennette and Hudson, Jour. Inf. Dis., *58*, 1936, 10–14; Loring and Schwerdt, Jour. Exp. Med., *75*, 1942, 395–406; McClure and Langmuir, Am. Jour. Hyg., *35*, 1942, 285–291; Melnick, Jour. Exp. Med., *77*, 1943, 195–204; Moore and Kessel, Am. Jour. Hyg., *38*, 1943, 323–344; Moore et al., *ibid.*, *36*, 1942, 247–254; Morales, Jour. Inf. Dis., *46*, 1930, 31–35; Olitsky and Cox, Jour. Exp. Med., *63*, 1936, 109–125; Paul et al., Am. Jour. Hyg., *17*, 1933, 587–600; 601–612; Jour. Exp. Med., *71*, 1940, 765–777; Sabin, *ibid.*, *69*, 1939, 507–516; Sabin and Olitsky, *ibid.*, *68*, 1938, 39–61; Sabin and Ward, *ibid.*, *73*, 1941, 771–793; *74*, 1942, 519–529; *75*, 1942, 107–117; Sabin et al., Jour. Bact., *31*, 1936, 35–36 (Abst.); Sanders and Jungeblut, Jour. Exp. Med., *75*, 1942, 631–649; Schultz and Gebhardt, Jour. Inf. Dis., *70*, 1942, 7–50; Schultz and Robinson, *ibid.*, *70*, 1942, 193–200; Stimpert and Kessel, Am. Jour. Hyg., *29*, (B), 1939, 57–66; Theiler, Medicine, *20*, 1941, 443–462; Theiler and Bauer, Jour. Exp. Med., *60*, 1934, 767–772; Trask and Paul, *ibid.*, *58*, 1933, 531–544; *73*, 1941, 453–459; Trask et al., *ibid.*, *77*, 1943, 531–544; Turner and Young, Am. Jour. Hyg., *37*, 1943, 67–79; Wolf, Jour. Exp. Med., *76*, 1942, 53–72; Young and Merrell, Am. Jour. Hyg., *37*, 1943, 80–92.

2. **Legio erebea** *spec. nov.* From Latin *erebeus*, belonging to the Lower World.

Common names: Choriomeningitis virus, lymphocytic choriomeningitis virus.

Hosts: *MURIDAE—Mus musculus* L., gray or white mouse. *HOMINIDAE—Homo sapiens* L., man. *CERCOPITHECIDAE—Macaca mulatta*, rhesus monkey. Experimentally, also guinea pig; white rat; dog (masked); ferret (masked); *Macaca irus*, crab-eating macaque; Syrian hamster; chick- or mouse-embryo serum-Tyrode solution culture; chick embryo.

Insusceptible species: Pig, rabbit, field vole, bank vole, canary, hen, parakeet.

Geographical distribution: France, England, United States.

Induced disease: In white mouse, more virulent in young than in old individuals; infection may take place *in utero* or soon after birth; some mice become carriers after recovery, with virus in organs, blood, urine, and nasal secretions; carriers are immune to large intracerebral inoculations of virus; experimentally, 5 to 12 days after intracerebral inoculation of susceptible mice, somnolence, photophobia, tremors of the legs, tonic spasms of muscles in the hindquarters upon stimulation; recovery or death. In man, disease may be subclinical at times as shown by the fact that some supposedly normal sera contain specific antibodies; not all clinical cases develop protecting antibodies against testing strains, so that disease may be somewhat commoner than can be ascertained readily; in all cases benign, but in the more severe of these an acute aseptic meningitis; after incubation period of 1½ to 3 days, spells of fever extending as long as 3 weeks; late in the disease there may be a meningeal reaction both clinically and cytologically; lymphocytes and some large mononuclear cells appear in the meningeal fluids, although symptoms remain benign; there may be virus in the blood from the beginning of fever to the end of the second week; the spinal fluid is not infective at first but may become so before there is a change in cell count; urine and saliva remain uninfectious.

Transmission: In white mouse, by contact with mice infected when young, not with those infected when old; nasal mucosa considered portal of entry. In wild gray mouse of the same species, *Mus musculus*, by contact but less readily than in white mouse. Experimentally, by mosquito, *Aëdes aegypti* L. (*CULICIDAE*), at 26 to 34° C; by bedbug, *Cimex lectularius* (*CIMIDAE*), but defecation on site of bitten area is essential, bite alone being ineffective. Experimentally, to guinea pig, by application of virus to normal and apparently intact skin; not by contamination of food or litter.

Serological relationships: Serum of recovered subjects usually neutralizes choriomeningitis virus. Hyperimmune serum is ineffective against pseudo-lymphocytic choriomeningitis virus and hyperimmune serum for that virus is ineffective in its turn when used with choriomeningitis virus. No cross neutralization with St. Louis encephalitis virus. A specific soluble antigen associated quantitatively with virus in all hosts fixes complement in the presence of immune serum; virus does so poorly if at all; the anti-soluble-substance antibodies seem to be independent of virus-neutralizing antibodies. A soluble protein, readily separable from virus, gives a specific precipitin reaction with immune serum; antibodies concerned are probably not the virus-neutralizing antibodies.

Immunological relationships: Intraperitoneal injection of about 160 intracerebral lethal doses has been found to protect the white mouse against infection by subsequent intracerebral injection of 10,000 lethal doses. The immune mouse differs from the immune guinea pig in showing no neutralizing antibodies in its blood; even the guinea pig may develop resistance before antibodies appear in its serum. Formalized vaccines made from

African monkey, by intracerebral, intranasal, and intraabdominal inoculation.

Serological relationships: Specific neutralizing antibodies arise after experimental infection in monkeys, but reinfection is not prevented; only a minority of human convalescent sera neutralize virus *in vitro*, the most potent sera probably being obtained from those with transient or light paralysis. Cross neutralization between monkey-passage and murine (cotton-rat and mouse) strains. No cross-neutralization reaction with lymphocytic choriomeningitis virus. Isolates differ somewhat antigenically, homologous titers being higher than heterologous titers in some neutralization tests.

Thermal inactivation: At or below 75° C in 30 minutes.

Filterability: Passes membrane about 35, not 30, millimicrons in average pore diameter.

Other properties: Infectivity of virus maintained well at −76° C or in glycerine but poorly when dried or just frozen. Inactivated readily by hydrogen peroxide. Particle diameter estimated as about 12 millimicrons by filtration studies. Precipitated by half-saturated ammonium sulphate solutions. Electron micrographs show elliptical particles 20 to 30 millimicrons in diameter; impure infectious materials show long threads 20 by 75 to 500 millimicrons in size. Component probably virus has sedimentation constant $S_{20^\circ} = 62 \times 10^{-13}$ cm per sec. per dyne. Inactivated by potassium hydroxide, copper sulfate and potassium permanganate. Stable from pH 2.2 to 10.4 for 2 hours at 37° C.

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Insusceptible species: Pig, rabbit, field vole, bank vole, canary, hen, parakeet.

Geographical distribution: France, England, United States.

Induced disease: In white mouse, more virulent in young than in old individuals; infection may take place *in utero* or soon after birth; some mice become carriers after recovery, with virus in organs, blood, urine, and nasal secretions; carriers are immune to large intracerebral inoculations of virus; experimentally, 5 to 12 days after intracerebral inoculation of susceptible mice, somnolence, photophobia, tremors of the legs, tonic spasms of muscles in the hindquarters upon stimulation; recovery or death. In man, disease may be subclinical at times as shown by the fact that some supposedly normal sera contain specific antibodies; not all clinical cases develop protecting antibodies against testing strains, so that disease may be somewhat commoner than can be ascertained readily; in all cases benign, but in the more severe of these an acute aseptic meningitis; after incubation period of 1½ to 3 days, spells of fever extending as long as 3 weeks; late in the disease there may be a meningeal reaction both clinically and cytologically; lymphocytes and some large mononuclear cells appear in the meningeal fluids, although symptoms remain benign; there may be virus in the blood from the beginning of fever to the end of the second week; the spinal fluid is not infective at first but may become so before there is a change in cell count; urine and saliva remain uninfectious.

Transmission: In white mouse, by contact with mice infected when young, not with those infected when old; nasal mucosa considered portal of entry. In wild gray mouse of the same species, *Mus musculus*, by contact but less readily than in white mouse. Experimentally, by mosquito, *Aëdes aegypti* L. (*CULICIDAE*), at 26 to 34° C; by bedbug, *Cimex lectularius* (*CIMIDAE*), but defecation on site of bitten area is essential, bite alone being ineffective. Experimentally, to guinea pig, by application of virus to normal and apparently intact skin; not by contamination of food or litter.

Serological relationships: Serum of recovered subjects usually neutralizes choriomeningitis virus. Hyperimmune serum is ineffective against pseudo-lymphocytic choriomeningitis virus and hyperimmune serum for that virus is ineffective in its turn when used with choriomeningitis virus. No cross neutralization with St. Louis encephalitis virus. A specific soluble antigen associated quantitatively with virus in all hosts fixes complement in the presence of immune serum; virus does so poorly if at all; the anti-soluble-substance antibodies seem to be independent of virus-neutralizing antibodies. A soluble protein, readily separable from virus, gives a specific precipitin reaction with immune serum; antibodies concerned are probably not the virus-neutralizing antibodies.

Immunological relationships: Intraperitoneal injection of about 160 intracerebral lethal doses has been found to protect the white mouse against infection by subsequent intracerebral injection of 10,000 lethal doses. The immune mouse differs from the immune guinea pig in showing no neutralizing antibodies in its blood; even the guinea pig may develop resistance before antibodies appear in its serum. Formalized vaccines made from

guinea pig tissues immunize the guinea pig but vaccines made from mouse tissues do not. Mice immune to this virus are susceptible to infection with pseudo-lymphocytic choriomeningitis virus and *vice versa*.

Thermal inactivation: At 55 to 56° C in 20 minutes.

Filterability: Passes Berkefeld V, N, and W filters and, with difficulty, a Seitz asbestos pad.

Other properties: Infective at least 206 days in storage at 4 to 10° C in 50 per cent neutral glycerine in 0.85 per cent saline. Infective particle calculated to be 37 to 55 millimicrons in diameter on the basis of centrifugation studies; 40 to 60 millimicrons by ultrafiltration tests. Inactivated by soap with loss of mouse-immunizing capacity.

Literature: Armstrong and Dickens, U. S. Pub. Health Service, Public Health Rept., *50*, 1935, 831–842; Armstrong and Lillie, *ibid.*, *49*, 1934, 1019–1027; Armstrong and Wooley, *ibid.*, *50*, 1935, 537–541; Jour. Am. Med. Assoc., *109*, 1937, 410–412; Baird and Rivers, Am. Jour. Pub. Health, *28*, 1938, 47–53; Casals-Ariet and Webster, Jour. Exp. Med., *71*, 1940, 147–154; Dalldorf, *ibid.*, *70*, 1939, 19–27; Dalldorf and Douglass, Proc. Soc. Exp. Biol. and Med., *39*, 1938, 294–297; Findlay and Stern, Jour. Path. and Bact., *43*, 1936, 327–338; Findlay et al., Lancet, *230*, 1936 (*I*), 650–654; Howard, Jour. Inf. Dis., *64*, 1939, 66–77; Laigret and Durand, Compt. rend. Acad. Sci., *203*, 1936, 282–284; Lépine and Sautter, Ann. Inst. Pasteur, *61*, 1938, 519–526; Lépine et al., *ibid.*, *204*, 1937, 1846–1848; MacCallum and Findlay, Brit. Jour. Exp. Path., *21*, 1940, 110–116; Milzer, Jour. Inf. Dis., *70*, 1942, 152–172; Rivers and Scott, Jour. Exp. Med., *63*, 1936, 415–432; Scott and Elford, Brit. Jour. Exp. Path., *20*, 1939, 182–188; Scott and Rivers, Jour. Exp. Med., *63*, 1936, 397–414; Shaugnessy and Zichis, *ibid.*, *72*, 1940, 331–343; Smadel and Wall, *ibid.*, *72*, 1940, 389–405; *75*, 1942, 581–591; Smadel et al., Proc. Soc. Exp. Biol. and Med., *40*, 1939, 71–73; Jour. Exp. Med., *70*, 1939, 53–66; *71*, 1940, 43–53; Stock and Francis, *ibid.*, *77*, 1943, 323–336; Traub, Science, *81*, 1935, 298–299; Jour. Exp. Med., *63*, 1936, 533–546, 847–861; *64*, 1936, 183–200; *66*, 1937, 317–324; *68*, 1938, 95–110, 229–250; *69*, 1939, 801–817.

3. **Legio simulans** *spec. nov.* From Latin *simulare*, to imitate, in reference to resemblance of this virus to the preceding in many respects, though not in size or antigenic properties.

Common name: Pseudo-lymphocytic choriomeningitis virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. Experimentally, also mouse, guinea pig, rhesus monkey; chorioallantoic membrane of chick embryo.

Induced disease: In man, benign aseptic lymphocytic meningitis with virus in cerebro-spinal fluid; severe frontal headache, drowsiness, irritability, vomiting, eventual complete recovery. In mouse, experimentally, roughened fur, spontaneous tremor, hunched attitude, irritability, clonic movements ending with tonic convulsions on stimulation, temporary recovery from spasm with survival a few hours or instant death.

Serological relationships: Hyperimmune sera for lymphocytic choriomeningitis virus are ineffective for this virus, and *vice versa*. In man, after recovery, neutralizing antibody is strong at 1 month, fading before 7 months.

Immunological relationships: Mice acquire specific resistance to reinfection after experimental disease; mice immune to lymphocytic choriomeningitis virus are susceptible to pseudo-lymphocytic choriomeningitis virus and *vice versa*.

Thermal inactivation: At 56° C, not at 45° C, in 30 minutes.

Filterability: Passes Berkefeld V, not N, filter candle; Gradacol membrane of 320, not 300, millimicron average pore diameter.

Other properties: Particle diameter calculated to be not above 150 to 225 millimicrons, from filtration experiments.

Viable at least 1 month at 4° C, at least 1 year in 50 per cent glycerine, 40 days in 0.25 per cent phenol, 1 year when dried from frozen material. Inactivated by 0.05 per cent formalin at 4° C in 48 hours; by boiling in 5 minutes.

Literature: MacCallum et al., Brit. Jour. Exp. Path., *20*, 1939, 260–269.

4. **Legio muris** *spec. nov.* From Latin *mus*, mouse.

Common names: Mouse-poliomyelitis virus, Theiler's-disease virus.

Host: *MURIDAE—Mus musculus* L., white mouse.

Insusceptible species: *CERCOPITHECIDAE—Macaca mulatta* (Zimmermann), rhesus monkey.

Geographical distribution: United States, Japan, Germany, Palestine; probably widespread wherever white mice are raised.

Induced disease: In white mouse, ordinarily no obvious disease, virus occurring in feces and not being recoverable from thoracic or abdominal viscera or head (probable source is in abdominal wall; virus has been recovered most abundantly from intestinal contents, in moderate amounts from walls of intestine and in smaller concentration from mesenteric lymph glands); occasionally, individual mice show flaccid paralysis of hind legs, and brain or spinal-cord suspensions from these contain the virus; mice inoculated intracerebrally show flaccid paralysis in 7 to more than 30 days, first in one limb, later usually in all; the tail does not become paralyzed; very young inoculated mice may die without first showing paralysis; very old inoculated mice may become infected without showing obvious disease; some affected mice recover and those showing residual paralysis may become carriers of virus. In affected, experimentally inoculated mice, acute necrosis of ganglion cells of anterior horn of spinal cord; necrosis also of isolated ganglion cells of cerebrum. Later, marked neuronophagia. Perivascular infiltration in brain and spinal cord. The reciprocal of the incubation period has been found approximately proportional to the logarithm of the amount of virus inoculated, thus serving to measure the concentration of samples of virus. Old mice less susceptible than young.

Transmission: Experimentally, by intracerebral, intranasal and intraperitoneal inoculation. Has been found to persist in adult flies, *Musca domestica* L. (*MUSCIDAE*) and other species, as long as 12 days after experimental feeding whereas mouse-adapted human poliomyelitis virus persists only 2 days in *Musca domestica* and not at all in some other species.

Serological relationships: Sera containing antibodies to the Lansing strain of human poliomyelitis virus fail to protect against mouse poliomyelitis virus.

Immunological relationships: Recovered mice are immune to various heterologous isolates or strains. No evidence of immunological relationship with virus of human poliomyelitis has been obtained, save that mice paralyzed with mouse poliomyelitis virus show some resistance to infection with the Lansing strain of human poliomyelitis virus; this has been interpreted as possibly no more than an interference phenomenon, since it seems to depend on actual paralysis.

Filterability: Passes Berkefeld N and other Berkefeld filters and Chamberland L_3 filter.

Other properties: Viable at least 14 months at −78° C; at least 150 days in 50 per cent glycerine at 2 to 4° C. Most stable near pH 8.0 and pH 3.3. Inactivated readily at 37° C by 1 per cent hydrogen peroxide. Particle diameter estimated as 9 to 13 millimicrons from filtration studies. Sedimentation constant, $S_{20°} = 160$ to 170×10^{-13} cm per sec. per dyne.

Literature: Bang and Glaser, Am. Jour. Hyg., *37*, 1943, 320–324; Gahagan and Stevenson, Jour. Inf. Dis., *69*, 1941, 232–237; Gard, Jour. Exp. Med., *72*, 1940, 69–77; Gard and Pedersen, Science, *94*, 1941, 493–494; Gildemeister and Ahlfeld,

Cent. f. Bakt., I Abt., Orig., *142*, 1938, 144–148; Iguchi, Kitasato Arch. Exp. Med., *16*, 1939, 56–78; Olitsky, Jour. Exp. Med., *72*, 1940, 113–127; Theiler, Science, *80*, 1934, 122; Jour. Exp. Med., *65*, 1937, 705–719; Theiler and Gard, *ibid.*, *72*, 1940, 49–67, 79–90; Young and Cumberland, Am. Jour. Hyg., *37*, 1943, 216–224.

5. **Legio gallinae** *spec. nov.* From Latin *gallina*, hen.

Common names: Avian encephalomyelitis virus, infectious avian encephalomyelitis virus.

Host: *PHASIANIDAE—Gallus gallus* (L.), chicken (embryo not susceptible; in culture media, minced whole embryo in serum-Tyrode solution suffices to maintain virus, but embryo brain alone does not).

Insusceptible species: All tested species other than birds.

Geographical distribution: United States.

Induced disease: In chicken, fine or coarse tremors of whole body or only of head and neck or of legs; progressive ataxia; eyes dull, some loss of weight, weakness of legs, and progressive incoordination of leg muscles; somnolence precedes death; about 75 per cent die within 5 days of onset, 90 per cent within a week, the remainder showing a staggering, ataxic gait for weeks, some continuously tremulous; recovered birds, however, may produce eggs well; microscopic focal collections of glia cells, perivascular infiltration, degeneration of Purkinje's cells and degeneration of nerve cells; foci of infiltration throughout brain and spinal cord; virus not detected in the blood of affected chickens.

Transmission: Not through egg. Experimentally, by intracerebral injection.

Serological relationships: Specific antiserum neutralizes homologous virus but not the Eastern strain of equine encephalitis virus; antiserum specific for the latter does not neutralize avian encephalomyelitis virus.

Filterability: Passes Berkefeld V and N as well as Seitz 1 and 2 filters; also membranes 73 millimicrons in average pore diameter.

Other properties: Survives in 50 per cent glycerine for at least 88 days and frozen for at least 68 days. Infective particle estimated to be 20 to 30 millimicrons in diameter, by filtration studies.

Literature: Jones, Science, *76*, 1932, 331–332; Jour. Exp. Med., *59*, 1934, 781–798; Kligler and Olitsky, Proc. Soc. Exp. Biol. and Med., *43*, 1940, 680–683; Olitsky, Jour. Exp. Med., *70*, 1939, 565–582; Olitsky and Bauer, Proc. Soc. Exp. Biol. and Med., *42*, 1939, 634–636; Van Roekel et al., Jour. Am. Vet. Med. Assoc., *93* (N.S. *46*), 1938, 372–375.

6. **Legio suariorum** *spec. nov.* From Latin *suarius*, swineherd.

Common name: Swineherds'-disease virus.

Hosts: *SUIDAE—Sus scrofa* L., swine. *HOMINIDAE—Homo sapiens* L., man. Experimentally, with fever as only symptom, white rat, cat, ferret, mouse; perhaps *Macaca mulatta* (Zimmermann), rhesus monkey.

Geographical distribution: Europe.

Induced disease: In man, a benign meningitis without sequelae, somewhat similar to lymphocytic choriomeningitis in man; cell counts in spinal fluids may be as high as 1200 to 1400; 4 to 7 (average 8) days after infection, fever lasting 3 to 21 days (average 9); sometimes conjunctivitis, more often a reddish maculopapillose eruption; severe sweating frequent; hemorrhagic tendency; blood in feces; recovery. Blood, urine, feces infectious, not spinal fluid or mucous excretions. Especially affecting young men, not often old men or women, among those having contact with swine or swine-producing quarters.

Transmission: Excreta of pigs, even as used for manure, are infective. Experimentally, to man, by subdermal or intramuscular injection.

Serological relationships: Serum from recovered cases neutralizes the virus.

Immunological relationships: Specific immunity follows attack of the disease.

Filterability: Passes Chamberland L_3 filter.

Literature: Durand et al., Compt. rend. Acad. Sci., Paris, *203*, 1936, 830–832, 957–959, 1032–1034; Arch. Inst. Pasteur de Tunis, *26*, 1937, 213–227; 228–249; *27*, 1938, 7–17.

Genus III. **Formido** *gen. nov.*

Viruses of the Rabies Group, inducing diseases characterized by involvement of the nervous system only. Generic name from Latin *formido*, a frightful thing.

The type and only recognized species is *Formido inexorabilis spec. nov.*

1. **Formido inexorabilis** *spec. nov.* From Latin *inexorabilis*, implacable.

Common name: Rabies virus.

Hosts: *CANIDAE—Canis familiaris* L., dog. *FELIDAE—Felis catus* L., domestic cat; *F. negripes*, black-footed cat; *F. ocreata*, wild cat. *HOMINIDAE—Homo sapiens* L., man. *MUSTELIDAE—Ictonyx orangiae*, polecat. *SCIURIDAE—Geosciurus capensis*, ground squirrel. *VIVERRIDAE—Cynictis penicillata*, yellow mongoose (yellow meercat); *Genetta felina* (Thunb.), genet cat; *Myonax pulverulentus*, small, grey mongoose; *Suricata suricatta*, Cape suricate or common meercat. Cattle, sheep, pig, horse, wolf. *Cynalopex chama*, silver jackal. *Phyllostoma superciliatum*, vampire bat; *Desmodus rufus*, vampire bat; *Artibeus planirostris trinitatis*, fruit-eating bat. Experimentally, also *Mus musculus* L., white mouse; *Peromyscus polionotus polionotus* (Wagner), white-footed mouse; tissue cultures of 5 or 6-day-old rat- or mouse-embryo brain; chick embryo (allantois not regularly infected, but virus regularly reaches brain of embryo without injuring it; chick may hatch with titer of 1:100 or 1:1000 in brain). Chicken; mouse hawk (*Buteo vulgaris*); pigeon, owl, goose; stork (*Ciconia ciconia*); pheasant (*Diardigallus diardi* B.P.).

Insusceptible species: Reptiles, fish. No mammal is known to be insusceptible.

Geographical distribution: Almost world-wide; absent only from relatively isolated countries or communities.

Induced disease: In dog, after a short incubation period (generally less than 10 days) altered behavior, hiding, lack of obedience, perverted appetite leading to ingestion of straw, paper, earth, and other unaccustomed materials; excitement, unprovoked biting (which may transmit the virus to new hosts), aimless wandering, excess salivation, progressive inability to swallow, alteration of bark to characteristic high pitched tone; staggering, paresis of hindquarters tending toward paralysis and involvement of anterior parts of the body; paralysis of lower jaw, muscular spasms, marked emaciation, death except perhaps in rare instances. In man, after a relatively long incubation period depending on site of implantation (perhaps 27 to 64 days), a uniformly fatal disease, characterized by altered behavior, increased excitability, thirst, pharyngeal spasm with progressive inability to swallow, labored and noisy respiration, death in 3 or 4 days after onset, with or without paroxysm. In sheep, increased sexual desire; tendency to pull wool from other sheep or themselves; light butting, increasing until some ewes, after violent exercise, appear to faint; prostration within 1 to 4 days; death within 2 days from onset of locomotory paralysis. In mouse, experimentally, by intracerebral inoculation, apathy, sluggishness, roughening of hair, tremor, convulsions, prostration, death; sometimes flaccid paralysis of hind legs before death.

Transmission: Usually by bite of dog or some closely related animal; occasionally by bites of cats; rarely by bites

of rabid horses or cattle. Not by contamination of food. In Brazil and Trinidad, probably by the vampire bat, which has been found infected in nature.

Serological relationships: Specific flocculation of rabies virus occurs in the presence of immune serum from rabbit or guinea pig; strains differ in relative amounts of antigenic constituents, as shown by absorption tests. Complement fixation occurs in the presence of virus and guinea-pig antiserum. Neutralizing antibodies are specific.

Immunological relationships: Virus exposed to ultraviolet light tends to lose its virulence before its immunizing potency. Passive immunization succeeds in white mice if antiserum is injected intracerebrally ½ hour before, but not 24 hours before or 2 hours after, virus. Chloroform-treated vaccines more effective than phenolized vaccines, but irritative.

Thermal inactivation: At 60 to 70° C in 15 minutes; in brain tissues, at 45° C in 24 hours.

Filterability: Passes Berkefeld V filter.

Other properties: Viable at least 2 months at 5° C in liquid or dry state. Infective particle between 100 and 240 millimicrons in diameter, by filtration studies.

Literature: Bernkopf and Kligler, Brit. Jour. Exp. Path., *18*, 1937, 481–485; Casals, Jour. Exp. Med., *72*, 1940, 445–451, 453–461; Covell and Danks, Am. Jour. Path., *8*, 1932, 557–572; Dawson, Science, *89*, 1939, 300–301; Am. Jour. Path., *17*, 1941, 177–188; Galloway, Brit. Jour. Exp. Path., *15*, 1934, 97–105; Goodpasture, Am. Jour. Hyg., *1*, 1925, 547–582; Haupt and Rehaag, Ztschr. f. Infektionskrankh., *22*, 1921, 76–88, 104–127; Havens and Mayfield, Jour. Inf. Dis., *50*, 1932, 367–376; *51*, 1932, 511–518; *52*, 1933, 364–373; Henderson, Vet. Med., *37*, 1942, 88–89; Hodes et al., Jour. Exp. Med., *72*, 1940, 437–444; Hoyt et al., Jour. Inf. Dis., *59*, 1936, 152–158; Hurst and Pawan, Lancet, *221*, 1931 (*2*), 622–628; Jour. Path. and Bact., *35*, 1932, 301–321; Johnson and Leach, Am. Jour. Hyg., *52* (B), 1940, 38–45; Kligler and Bernkopf, Proc. Soc. Exp. Biol. and Med., *39*, 1938, 212–214; Am. Jour. Hyg., *33* (B), 1941, 1–8; Leach and Johnson, *ibid.*, *32* (B), 1940, 74–79; Metivier, Jour. Comp. Path. and Therap., *48*, 1935, 245–260; Peragallo, Giorn. di batteriol. e immunol., *18*, 1937, 289–290; Snyman, Onderstepoort Jour. Vet. Sci. and Anim. Indust., *15*, 1940, 9–140; Webster, Am. Jour. Pub. Health, *26*, 1936, 1207–1210; Jour. Exp. Med., *70*, 1939, 87–106; Am. Jour. Hyg., *30* (B), 1939, 113–134; Webster and Casals, Jour. Exp. Med., *71*, 1940, 719–730; *73*, 1941, 601–615; *76*, 1942, 185–194; Webster and Clow, *ibid.*, *66*, 1937, 125–131; Wyckoff, Am. Jour. Vet. Res., *2*, 1941, 84–90.

Note: The Negri body, a characteristic cell-inclusion in rabies, has been given the following names under the supposition that it represents stages in the life cycle of a protozoan parasite responsible for the disease: *Neuroryctes hydrophobiae* by Calkins, Jour. Cutaneous Diseases including Syphilis, *25*, 1907, 510; *Encephalitozoon rabiei* by Manouelian and Viala, Ann. Inst. Pasteur, *38*, 1924, 258; and *Glugea lyssae* by Levaditi, Nicolau and Schoen, Ann. Inst. Pasteur, *40*, 1926, 1048.

FAMILY IV. CHARONACEAE *FAM. NOV.*

Viruses of the Yellow-Fever Group, inducing diseases mainly characterized by fever and necrosis of tissues in the absence of obvious macule, papule, or vesicle formation or of conspicuous involvement of nerve cells.

Key to the genera of family **Charonaceae.**

I. Viruses of the Typical Yellow-Fever Group.
Genus I. *Charon*, p. 1265.
II. Viruses of the Influenza Group.
Genus II. *Tarpeia*, p. 1268.
III. Viruses of the Hog-Cholera Group.
Genus III. *Tortor*, p. 1275.

Genus I. Charon *gen. nov.*

Viruses of the Typical Yellow-Fever Group, inducing diseases mainly characterized by acute non-contagious fever. Vectors dipterous insects, so far as known. Generic name from Latin *Charon*, ferryman of the Lower World.

The type species is *Charon evagatus spec. nov.*

Key to the species of genus **Charon.**

I. Vectors mosquitoes.
1. *Charon evagatus.*
II. Vectors unknown, perhaps mosquitoes.
2. *Charon vallis.*

1. **Charon evagatus** *spec. nov.* From Latin *evagor*, to spread abroad.

Common name: Yellow-fever virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. Experimentally, also *Cercopithecus tantalus* Ogilby; *C. aethiops*, African guenon (symptomless); *Cercocebus torquatus* (Kerr), collared mangabey; *Mus musculus* L., mouse; *Microtus agrestis*, field vole; *Sciurus vulgaris* L., red squirrel; *Macaca mulatta* (Zimmermann), rhesus monkey; *Macacus sinicus* Indian crown monkey; *M. cynomolgus*; *M. speciosus*; *Erinaceus europaeus*, hedge hog; *Gallus gallus* (L.), chicken (tolerant); *Dasyprocta aguti*, agouti (serial passage fails).

Insusceptible species: Cat, ferret, rabbit, rat; *Cricetus auratus*, golden hamster; *Apodemus sylvaticus*, wood vole; *Evotomys glareolus*, bank vole; pigeon, canary, pipistrelle bat; *Cricetomys gambianus*, pouched rat; dog, goat.

Geographical distribution: Tropical regions in general, especially Central and South America, West Indies, West Africa; anti-mosquito campaigns have tended to eradicate yellow-fever virus from parts of its former range.

Induced disease: In man, mild cases may occur, especially in natives where the disease is endemic, but in Europeans generally sudden fever without marked change in pulse rate after a 3 to 6-day incubation period; severe frontal headache, pains in the loin and legs and epigastric pain; gradual decrease in temperature to 98 or 99° F, weakening of pulse and slowing of heart beat in the absence of further temperature changes; jaundice, especially in sclerae, often in skin; albumen in urine, later bile-pigments also present; hemorrhages frequent especially in alimentary canal; fatty and necrotic changes in the liver; acute degeneration of renal parenchyma, splenic congestion; death may occur in the early acute state, but is more likely about the

fifth or sixth day; relapses may occur until 2 or 3 weeks after onset; case mortality varies from 10 to 90 per cent in different epidemics. A transitory immunity due to transfer of serum antibodies through the placenta protects offspring of immune mothers for a short time.

Transmission: By mosquitoes, *Aëdes aegypti* L., *Aëdes leucocelaemus* (D. and S.), *Haemogogus capricorni* Lutz (*CULICIDAE*). The mosquito *Aëdes aegypti* becomes infective, after feeding on a suitable virus source, in 4 days at 37° C, 5 days at 36° C, 6 days at 31° C, 8 days at 25.1° C, 9 to 11 days at 23.4° C, 18 days at 21° C, and 36, not 30, days at 18° C; virus in head, thorax, and abdomen before bites are infective; no evidence of transmission of virus through eggs to offspring or to larvae eating infected adults. Experimentally, also by *Aëdes scapularis* (Rondani), *A. fluviatilis* (Lutz), *A. luteocephalus*, *A. apico-annulatus* (*CULICIDAE*). Experimentally, by feeding, to *Macaca mulatta* and *Cercopithecus aethiops*; by rubbing infected blood into intact and unshaved skin of monkeys.

Serological relationships: Complement-fixation and precipitating antibodies are specific.

Immunological relationships: A specific immunity develops after an attack of the disease or after vaccination with virus grown in media containing tissues of chick embryo minus head and spinal cord.

Thermal inactivation: At 55 to 60° C, not at 50° C, in 10 minutes.

Filterability: Passes membranes of 55, and to some extent membranes of 50, millimicron average pore diameter. Passes Berkefeld V and N, as well as Chamberland F, filters.

Other properties: Particle estimated from filtration data to have a diameter of 17 to 28 millimicrons; by ultracentrifugation data, 19 millimicrons. Inactivated or inhibited by 30-minute exposure to 1:15 formalin, 1:6 ethyl alcohol; 1:300 yellowish eosin, 1:50 sodium oleate, 1:200 *liquor cresolis compositus*; viable after 30-minute exposure at 30° C to 1:7500 mercuric chloride, 1:150 phenol, 1:1500 hexylresorcinol, 1:150 sodium oleate. Sedimentation constant between 18 and 30×10^{-13} cm per sec. per dyne. Viable in 50 per cent glycerine at 2 to 4° C for 58, not for 100, days; in mouse brain at −8° C for 160 days. Viability may be lost on simple drying but retained if drying is carried on *in vacuo* over a desiccating agent.

Strains: Distinctive strains have been isolated. One, to which much study has been given, differs from the typical viscerotropic strain by possessing marked neurotropic or pantropic characteristics.

Literature: Bauer and Hughes, Am. Jour. Hyg., *21*, 1935, 101–110; Bauer and Mahaffy, *ibid.*, *12*, 1930, 155–174; 175–195; Bugher and Gast-Galvis, *ibid.*, *39*, 1944, 58–66; Bugher et al., *ibid.*, *39*, 1944, 16–51; Davis, *ibid.*, *16*, 1932, 163–176; Davis and Shannon, *ibid.*, *11*, 1930, 335–344; Davis et al., Jour. Exp. Med., *58*, 1933, 211–226; Findlay, Jour. Path. and Bact., *38*, 1934, 1–6; Lancet, *227*, 1934 (2), 983–985; Findlay and Clarke, Jour. Path. and Bact., *40*, 1935, 55–64; Findlay and MacCallum, Brit. Jour. Exp. Path., *19*, 1938, 384–388; Jour. Path. and Bact., *49*, 1939, 53–61; Findlay and Mackenzie, *ibid.*, *43*, 1936, 205–208; Findlay and Stern, *ibid.*, *40*, 1935, 311–318; Fox and Cabral, Am. Jour. Hyg., *37*, 1943, 93–120; Frobisher, Am. Jour. Hyg., *11*, 1930, 300–320; *13*, 1931, 585–613; *14*, 1931, 147–148; *18*, 1933, 354–374; Goodpasture, Am. Jour. Path., *8*, 1932, 137–150; Haagen, Deutsch. med. Wochnschr., *60*, 1934, 983–988; Hudson, Am. Jour. Path., *4*, 1928, 395–430; Klotz and Simpson, *ibid.*, *3*, 1927, 483–488; Laemmert and Moussatché, Jour. Inf. Dis., *72*, 1943, 228–231; Lloyd et al., Am. Jour. Hyg., *18*, 1933, 323–344; Trans. Roy. Soc. Trop. Med. and Hyg., *29*, 1936, 481–529; Mahaffy et al., Am. Jour. Hyg., *18*, 1933, 618–628; Pickels and Bauer, Jour. Exp. Med., *71*, 1940,

703–717; Ramsey, Am. Jour. Hyg., *13*, 1931, 129–163; Sawyer, *ibid.*, *25*, 1937, 221–231; Shannon et al., Science, *88* 1938, 110–111; Smith and Theiler, Jour. Exp. Med., *65*, 1937, 801–808; Smith et al., Am. Jour. Trop. Med., *18*, 1938, 437–468; Soper and De Andrade, Am. Jour. Hyg., *18*, 1933, 588–617; Soper et al., *ibid.*, *18*, 1933, 555–587; *19*, 1934, 549–566; *27*, 1938, 351–363; Stefanopoulo and Wassermann, Bull. Soc. Path. Exot., *26*, 1933, 557–559; Stokes et al., Am. Jour. Trop. Med., *8* 1928, 103–164; Theiler, Ann. Trop. Med. and Parasit., *24*, 1930, 249–272; Theiler and Smith, Jour. Exp. Med., *65*, 1937, 767–786, 787–800; Whitman, *ibid.*, *66*, 1937, 133–143.

2. **Charon vallis** *spec. nov.* From Latin *vallis*, valley.

Common name: Rift Valley fever virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. *BOVIDAE—Bos taurus* L., cow; *Ovis aries* L., sheep; *Capra hircus* L., goat. Experimentally, also *Sciurus carolinensis*, grey squirrel; ferret; *Cricetus auratus*, golden hamster; *Apodemus sylvaticus*, wood mouse; *Microtus agrestis* field vole; *Muscardinus avellanarius*, dormouse; rat; mouse; *Macaca mulatta*; *M. irus*; *Cebus fatuellus*; *C. chrysopus*; *Hapale jacchus*; *H. penicillata*; *Cercopithecus callitrichus* (symptomless); *Erythrocebus patas* (symptomless); *Cercocebus fuliginosus* (symptomless); chick embryo in Tyrode's solution; chorioallantoic membrane of chick embryo.

Insusceptible species: Horse, pig.

Geographical distribution: British East Africa.

Induced disease: In man, benign disease; after 5½ to 6 days, rigors, pains in back, fever for 12 to 36 hours, followed by recovery, with persistence of acquired immune bodies as long as 4 to 5 years after infection. In sheep (lambs), dullness, rapid respiration, collapse and death in a few hours or a chronic course; focal necrosis in liver. In chorioallantoic membrane of chick embryo, experimentally, areas of hyperplasia and of necrosis; connective tissue inflamed nearby; liver of embryo mottled with necrotic areas.

Transmission: Not by contacts. Mosquito, *Taeniorhynchus brevipalpis* (*CULICIDAE*), suspected as possible vector.

Serological relationships: Antisera for psittacosis, dengue fever, and sandfly fever viruses fail to protect against infection with Rift Valley fever virus. Specific neutralizing antibody in intraperitoneally neutral mixture with Rift Valley fever virus may be dissociated so as to free virus by direct dilution in saline solutions, by intranasal inoculation, or by employment of a small dose, all methods probably implying a dilution effect.

Immunological relationships: No cross immunity with yellow-fever or dengue-fever viruses. If Rift Valley fever virus is inoculated into *rhesus* monkey simultaneously with yellow-fever virus, the animal tends to be protected against death from yellow fever (interference effect), but one-day earlier inoculation of Rift Valley fever virus does not protect.

Thermal inactivation: At 56° C in 40, not 20, minutes.

Filterability: Passes Berkefeld V, N, and W filters; passes Chamberland L_2, L_3, L_5, L_7, L_{11} and occasionally L_{13} filters; passes membranes 150 millimicrons in average pore diameter freely, 90 millimicrons with difficulty, 70 millimicrons not at all.

Other properties: Viable at least 8 months at 4° C, more than 4 weeks dry in liver tissues, 6 months in ½ per cent carbolic acid at 4° C. Diameter of infective particle estimated from filtration studies to be between 23 and 35 millimicrons.

Strains: A neurotropic strain immunizes lambs without producing obvious illness, if given subcutaneously.

Literature: Broom and Findlay, Brit. Jour. Exp. Path., *14*, 1933, 179–181; Daubney et al., Jour. Path. and Bact., *34*, 1931, 545–579; Findlay, Trans. Roy.

Soc. Trop. Med. and Hyg., *25*, 1932, 229–266; *26*, 1932, 157–160; 161–168; Brit. Jour. Exp. Path., *17*, 1936, 89–104; Findlay and MacCallum, Jour. Path. and Bact., *44*, 1937, 405–424; Findlay and Mackenzie, Brit. Jour. Exp. Path., *17*, 1936, 441–447; Findlay et al., *ibid.*, *17*, 1936, 431–441; Francis and Magill, Jour. Exp. Med., *62*, 1935, 433–448; Horning and Findlay, Jour. Roy. Micr. Soc., *54*, 1934, 9–17; Mackenzie, Jour. Path. and Bact., *37*, 1933, 75–79; *40*, 1935, 65–73; Mackenzie et al., Brit. Jour. Exp. Path., *17*, 1936, 352–361; Saddington, Proc. Soc. Exp. Biol. and Med., *31*, 1934, 693–694; Schwentker and Rivers, Jour. Exp. Med., *59*, 1934, 305–313.

Genus II. **Tarpeia** *gen. nov.*

Viruses of the Influenza Group, inducing diseases characterized principally by involvement of the respiratory tract. Generic name from Latin *Tarpeia*, name of a Roman maiden who treacherously opened a citadel to an enemy.

The type species is *Tarpeia alpha spec. nov.*

Key to the species of genus **Tarpeia.**

I. Infecting man principally.
   1. *Tarpeia alpha.*
   2. *Tarpeia beta.*
   3. *Tarpeia premens.*

II. Affecting feline species.
   4. *Tarpeia felis.*

III. Affecting domestic cattle (calves).
   5. *Tarpeia vitulae.*

IV. Affecting canine species.
   6. *Tarpeia canis.*
   7. *Tarpeia vulpis.*

V. Affecting ferrets.
   8. *Tarpeia viverrae.*

VI. Affecting domestic fowl.
   9. *Tarpeia avium.*

1. **Tarpeia alpha** *spec. nov.* From first letter of Greek alphabet.

Common name: Influenza A virus; swine filtrate-disease virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. *SUIDAE—Sus scrofa* L., domestic swine. Experimentally, also ferret, mouse, *Macacus irus*, hedgehog, rabbit (inapparent infection), guinea pig (inapparent infection), rat (inapparent infection); *Mustela sibirica* Milne-Edwards, Chinese mink; *Sciurotamius davidianus* Milne-Edwards, David's squirrel; chick embryo (some strains produce visible lesions at 36.5° C on chorioallantoic membrane); minced chick embryo in Tyrode's solution.

Insusceptible species: *Callosciurus caniceps canigenus* Howell, Chekiang squirrel; *Eutamias asiaticus senescens* Miller, chipmunk.

Geographical distribution: World-wide.

Induced disease: In man, headache, dizziness, with shivering and muscular pains; rise of temperature on the second day, sometimes with fall on the third and elevation again later; often complicated by bronchitis and bronchopneumonia; hemorrhagic and edematous lobular consolidation in lungs; virus most easily recoverable from nasopharyngeal washings, but also from nasal secretions and lungs. In swine, virus alone produces only a mild malady (filtrate disease);

in the presence of *Haemophilus influenzae suis* a severe malady occurs under both natural and experimental conditions; it involves fever, cough, and prostration; many infected animals die. Lungworms, *Metastrongylus elongatus* and *Choerostrongylus pudendotectus* (*METASTRONGYLIDAE*), from infected swine harbor virus at least 2 years, living meantime in earthworms, such as *Allolobophora caliginosa* (*LUMBRICIDAE*), which are eaten eventually by swine. The swine are refractory to viral infection during May, June, July, and August, but the disease may be invoked later by successive intramuscular injections of *Haemophilus influenzae suis* or other stimuli, such as feeding embryonated *Ascaris* ova. In infected swine, virus occurs in turbinates, tracheal exudates, and lungs; not in spleen, liver, kidney, mesenteric lymph nodes, brain, blood, or mucosa of colon. Neutralizing antibodies appear later (7th to 10th day) in the mild filtrate disease than in typical swine influenza, in which they appear about the 6th to the 7th day; maximum titer on 14th to 27th day. Experimentally in mouse, not contagious as in swine and not dependent on the coexistence of a bacterial component; death of epithelium of respiratory and terminal bronchioles, complete epithelial desquamation, dilatation of bronchioles, collapse of alveoli; in healing, widespread epithelial proliferation. Experimentally in ferret, moderate apathy, lack of appetite, pallor of nose, variable catarrhal symptoms; at acute stage of disease, necrosis of respiratory epithelium of nasal mucous membrane, with desquamation of superficial cells, exudation into air passages and inflammatory reaction in the submucosa; repair follows, beginning on the 6th day after infection and becoming essentially complete at the end of 1 month; after recovery, the ferret is immune for 3 months or more, with subsequent waning of resistance; subsequent subcutaneous inoculations of virus restore immunity.

Transmission: Presumably by droplets; for example between cages of ferrets as close as 5 feet apart, even to levels 3 feet higher than cage of diseased individuals. Experimentally, from washings of human throats to ferret, mouse, chick embryo (by amniotic route and to allantoic membrane); in mice, by contact and by inhalation of fine droplets.

Serological relationships: Neutralizing antibodies common in human sera from individuals above 10 years of age; rarer in sera from young children; strongly effective for homologous, weak for heterologous, virus in convalescent sera. Soluble complement-fixing antigen of swine strain has components in common with antigens of human strains (PR8 and WS). Complement fixation best 10 to 14 days after onset in man. Inactivating capacity of nasal secretions proportional to level of neutralizing antibodies in blood. Agglutination of red cells by influenza virus is inhibited quantitatively by specific antiserum.

Immunological relationships: Specific immunization of ferrets, without obvious disease, occurs as a result of intranasal inoculation of egg-passage influenza virus that is not transmissible from ferret to ferret. In mice, immunizing dose is directly proportional to degree of induced immunity; immunity to the strain used in immunization is more effective in general than that to heterologous isolates of the virus.

Filterability: Passes Berkefeld V filter.

Other properties: Particle size estimated as 80 to 120 millimicrons by filtration studies; 80 to 99 millimicrons by ultracentrifugation ($S_{20^\circ} = 724 \times 10^{-13}$ cm per sec. per dyne); electron micrographs show bean or kidney-shaped particles, or round particles with central dense spot, averaging 77.6 millimicrons in diameter. Inactivated by oleic, linolic and linolenic acids without loss of immunizing ability. Inactivated by ultraviolet radiation.

Literature: Andrewes and Glover,

Brit. Jour. Exp. Path., *22*, 1941, 91–97; Andrewes et al., *ibid.*, *16*, 1935, 566–582; Burnet, *ibid.*, *17*, 1936, 282–293; *18*, 1937, 37–43; *21*, 1940, 147–153; Austral. Jour. Exp. Biol. and Med. Sci., *14*, 1936, 241–246; *19*, 1941, 39–44, 281–290; Eaton, Jour. Bact., *39*, 1940, 229–241; Eaton and Pearson, Jour. Exp. Med., *72*, 1940, 635–643; Eaton and Rickard, Am. Jour. Hyg., *33*, (B), 1941, 23–35; Elford et al., Brit. Jour. Exp. Path., *17*, 1936, 51–53; Francis, Science, *80*, 1934, 457–459; Jour. Exp. Med., *69*, 1939, 283–300; Francis and Magill, Science, *82*, 1935, 353–354; Brit. Jour. Exp. Path., *19*, 1938, 284–293; Francis and Shope, Jour. Exp. Med., *63*, 1936, 645–653; Francis and Stuart-Harris, *ibid.*, *68*, 1938, 789–802; Francis et al., Am. Jour. Hyg., *37*, 1943, 294–300; Hirst, Jour. Exp. Med., *75*, 1942, 49–64; Hirst et al., *ibid.*, *75*, 1942, 495–511; Proc. Soc. Exp. Biol. and Med., *50*, 1942, 129–133; Horsfall and Lennette, Jour. Exp. Med., *73*, 1941, 327–333; Hudson et al., *ibid.*, *77*, 1943, 467–471; Hyde, Am. Jour. Hyg., *36* 1942, 338–353; Lennette and Horsfall, Jour. Exp. Med., *73*, 1941, 581–599; Loosli et al., Jour. Inf. Dis., *72*, 1943, 142–153; Lush and Burnet, Austral. Jour. Exp. Biol. and Med. Sci., *15*, 1937, 375–383; Magill and Francis, Brit. Jour. Exp. Path., *19*, 1938, 273–284; Nigg et al., Am. Jour. Hyg., *34* (B), 1941, 138–147; Orcutt and Shope, Jour. Exp. Med., *62*, 1935, 823–826; Rosenbusch and Shope, *ibid.*, *69*, 1939, 499–505; Shope, *ibid.*, *59*, 1934, 201–211; *62*, 1935, 561–572; *64*, 1936, 47–61; *67*, 1938, 739–748; *74*, 1941, 41–47, 49–68; *77*, 1943, 111–126, 127–138; Shope and Francis, *ibid.*, *64*, 1936, 791–801; Smillie, Am. Jour. Hyg., *11*, 1930, 392–398; Smith et al., Lancet, *225*, 1933 (*2*), 66–68; Brit. Jour. Exp. Path., *16*, 1935, 291–302; Smorodintseff and Ostrovskaya, Jour. Path. and Bact., *44*, 1937, 559–566; Stock and Francis, Jour. Exp. Med., *71*, 1940, 661–681; Straub, Jour. Path. and Bact., *45*, 1937, 75–78; Stuart-Harris, Brit. Jour. Exp. Path., *17*, 1936, 324–328; *18*, 1937, 485–492; Sulkin et al., Jour. Inf. Dis., *69*, 1941, 278–284; Tang, Brit. Jour. Exp. Path., *19*, 1938, 179–183; Taylor, (A. R.), et al., Jour. Immunol., Virus Res. and Exp. Chemother., *47*, 1943, 261–282; Taylor, (R. M.), et al., Am. Jour. Hyg., *31*, (B), 1940, 36–45; Jour. Inf. Dis., *68*, 1941, 90–96; Wells and Brown, Am. Jour. Hyg., *24*, 1936, 407–413.

2. **Tarpeia beta** *spec. nov.* From second letter of Greek alphabet.

Common name: Influenza B virus.

Hosts: *HOMINIDAE—Homo sapiens* L., man. Experimentally, also ferret, mouse, chick embryo.

Geographical distribution: United States, England.

Induced disease: In man, subclinical disease or one resembling that induced by influenza A virus. In chick embryo, experimentally, virus increases in entodermal cells lining allantoic cavity.

Serological relationships: Not neutralized by antiserum to influenza A virus. Specific neutralization and complement-fixation reactions. Rapidly adsorbed by normal chicken-blood red cells (95 per cent in 15 minutes); released in 4 hours essentially completely; the process is then repeatable with fresh red cells.

Other properties: Particle circular or bean-shaped in outline, with average diameter of 97.3 millimicrons in electron micrographs; of 99.8 millimicrons by centrifugation studies.

Literature: Burnet, Austral. Jour. Exp. Biol. and Med. Sci., *19*, 1941, 291–295; Francis, Science, *92*, 1940, 405–408; Proc. Soc. Exp. Biol. and Med., *45*, 1940, 861–863; Hirst, Jour. Exp. Med., *76*, 1942, 195–209; Lush et al., Brit. Jour. Exp. Path., *22*, 1941, 302–304; Nigg et al., Am. Jour. Hyg., *35*, 1942, 265–284; Sharp et al., Jour. Immunol., Virus Res. and Exp. Chemother., *48*, 1944, 129–153.

3. **Tarpeia premens** *spec. nov.* From Latin *premere*, to oppress or afflict.

Common name: Common-cold virus.

Hosts: *HOMINIDAE—Homo sapiens*

L., man. Experimentally, also chimpanzee, chick embryo.

Geographical distribution: World-wide except in conditions of isolation of small communities.

Induced disease: In man, incubation period about 48 hours; mild malady; running nose in 81 per cent of cases, obstruction of nostrils in 44 per cent, sudden onset in 37 per cent, cough in 31 per cent, headache in 19 per cent, sore throat in 14 per cent, fever in 13 per cent, inflammation of eyes in 12 per cent; changes in weather, especially during a warm season, predispose to the disease; no correlation between susceptibility and outdoor exercise, exposure to fresh air while sleeping, eye color, adenotonsillectomy, or size of frontal sinus. Incidence inversely proportional to daily hours of sunshine and atmospheric temperature. Fitness (defined by speed of oxygen replacement) correlated with relative freedom from colds. Effect of rest during disease favorable, reducing complications, length of fever, duration of illness, and period off duty.

Immunological relationships: After attack, specific immunity for about 7 weeks (minimum period 23 days); then exposure to chilling may cause a relapse, but an isolated community tends to lose the virus during the refractory period.

Filterability: Passes Berkefeld V and W as well as Seitz filters.

Other properties: Viable at least 13 days at ice-box temperature, anaerobically; at least 4 months frozen and dried *in vacuo*. Gum acacia tends to stabilize virus in chick-embryo tissue medium.

Literature: Dochez et al., Jour. Exp. Med., *63*, 1936, 559–579; Doull et al., Am. Jour. Hyg., *13*, 1931, 460–477; *17*, 1933, 536–561; Gafafer, *ibid.*, *13*, 1931, 771–780; *16*, 1932, 233–240, 880–884; Jour. Inf. Dis., *51*, 1932, 489–492; Gafafer and Doull, Am. Jour. Hyg., *18*, 1933, 712–726; Hyde and Chapman, *ibid.*, *26*, 1937, 116–123; Kneeland et al., Proc. Soc. Exp. Biol. and Med., *35*, 1936, 213–215; Le Blanc and Welborn, Am. Jour. Hyg., *24*, 1936, 19–24; Locke, Jour. Inf. Dis., *60*, 1937, 106–112; Long and Doull, Proc. Soc. Exp. Biol. and Med., *28*, 1930, 53–55; Maughan and Smiley, Am. Jour. Hyg., *9*, 1929, 466–472; Noble and Brainard, Jour. Bact., *29*, 1935, 407–409; Palmer, Am. Jour. Hyg., *16*, 1932, 224–232; Paul and Freese, *ibid.*, *17*, 1933, 517–535; Shibley et al., Jour. Am. Med. Assoc., *95*, 1930, 1553–1556; Smiley, Am. Jour. Hyg., *6*, 1926, 621–626; *9*, 1929, 477–479.

4. **Tarpeia felis** *spec. nov.* From Latin *feles*, cat.

Common name: Feline-distemper virus.

Hosts: *FELIDAE—Felis catus* L., domestic cat; *F. pardus*, leopard; *F. tigrina*, American tiger cat; *F. aurata*, African tiger cat; *F. planiceps*, rusty tiger cat; *F. marmorata*, marbled cat; *F. caracal*, caracal lynx; *F. pardalis*, ocelot; lion, tiger, puma relatively insusceptible.

Insusceptible species: Man, dog, ferret, mongoose, rabbit, rat, mouse, guinea pig.

Induced disease: In domestic cat, coughing, sneezing, running eyes and nose, with serous or purulent conjunctivitis, or diarrhea and vomiting; fever to 103 or 105° F; loss of appetite, general weakness; mortality high, especially among young individuals; death usually occurs on the 10th to the 12th day, in extreme cases, however, as early as the 5th or as late as the 35th day; catarrhal congestion in some part of the gastrointestinal tract is typical; this ranges from a few small patches in the ileum to involvement of the whole small intestine and parts of the large intestine or stomach and esophagus; often enlargement and congestion of abdominal lymph glands, enlargement of spleen, pleurisy, and peritonitis.

Filterability: Passes Berkefeld N and Chamberland L_3 filters.

Transmission: By fomites.

Immunological relationships: Recovered cats specifically immune.

Other properties: Viable at least 3 weeks in 50 per cent glycerine; attenuated or killed by drying at room temperature, but some immunization is reported if dried virus is injected.

Literature: Dalling, Vet. Record, *15*, 1935, 283-289; Findlay, Vet. Jour., *89*, 1933, 17-20; Hindle and Findlay, Jour. Comp. Path. and Therap., *45*, 1932, 11-26; Verge and Cristoforoni, Compt. rend. Soc. Biol., Paris, *99*, 1928, 312-314.

5. **Tarpeia vitulae** *spec. nov.* From Latin *vitula*, cow-calf.

Common name: Pneumoenteritis virus

Hosts: *BOVIDAE—Bos taurus* L., domestic cattle. Experimentally, also *MURIDAE—Mus musculus* L., mouse.

Geographical distribution: United States.

Induced disease: In cattle (calves), after incubation period of 2 to 4 days, fever increasing rapidly to 40 or 41° C and lasting 3 to 5 days; usually after first day of fever, diarrhea with feces soft, yellow, voluminous, fetid in odor, occasionally blood-tinged or fluid; diarrhea is followed by pneumonia and recovery after disappearance of fever; catarrhal enteritis and a bronchopneumonia usually confined to the anterior lobes of the lungs underlie the symptoms; no inclusion bodies in cells of affected tissues.

Transmission: By pen contacts with infected calves. Experimentally, by intranasal inoculation of calves, using inocula prepared from lungs of infected mice.

Serological relationships: Recovered animals develop neutralizing antibodies.

Immunological relationships: A specific resistance to reinfection is conferred by an attack of the disease.

Filterability: Passes Berkefeld N filter.

Literature: Baker, Cornell Vet., *32*, 1942, 202-204; Jour. Exp. Med. *78*, 1943, 435-446.

6. **Tarpeia canis** *spec. nov.* From Latin *canis*, dog.

Common name: Canine-distemper virus.

Hosts: *CANIDAE—Canis familiaris* L., dog; *Vulpes* sp., fox. *MUSTELIDAE*—ferret.

Insusceptible species: Man, rabbit, guinea pig, white rat, cat.

Geographical distribution: Widespread throughout the world.

Induced disease: In dog, after 4 days from time of infection, fever and a watery discharge from the eyes and nose, sometimes inconspicuous but often profuse; usually diarrhea and wasting followed by recovery or, exceptionally, death. Virus passes from the respiratory tract through the blood stream to its favored sites in vascular endothelium and cells of the reticulo-endothelial system. Nuclear inclusions are found in liver cells, bronchial epithelial cells, glandular cells of the stomach and intestine, and bile-duct epithelial cells; there are also cytoplasmic inclusions in bile-duct epithelial cells.

Transmission: By contact. Probably by air-borne droplets. No arthropod vector is recognized.

Immunological relationships: Dead-vaccine treatment followed by living-virus treatment produces a lasting immunity. Virus inactivated by photodynamic effect in 2 mm layer of 1:50,000 or 1:100,000 methylene blue, exposed 30 minutes at 20 cm from 100 candle-power lamp, still immunizes. Vaccine may be dried.

Filterability: Passes Chamberland L_3 and Mandler filters.

Other properties: Viable in liver tissue at 10° C for 35, not 85, days; in glycerine-saline solution at 10° C, 67 days though deteriorated; in vacuum-dried liver tissue, at 10° C, 90 days. If dried from frozen state, virus is viable in vacuum at least 430 days at 7° C, in oxygen-free nitrogen at least 365 days at 7° C. Viable in 25 per cent sterile horse serum at −24° C more than 693 days.

Literature: Carré, Compt. rend. Acad. Sci., Paris, *140*, 1905, 689-690; Dalldorf,

Jour. Exp. Med., *70*, 1939, 19–27; De Monbreun, Am. Jour. Path., *18*, 1937, 187–212; Dunkin and Laidlaw, Jour. Comp. Path. and Therap., *39*, 1926, 201–212, 213–221; Green and Evans, Am. Jour. Hyg., *29* (B), 1939, 73–87; Laidlaw and Dunkin, Jour. Comp. Path. and Therap., *39*, 1926, 222–230; *41*, 1928, 1–17, 209–227; Perdrau and Todd, *ibid.*, *46*, 1933, 78–89; Siedentopf and Green, Jour. Inf. Dis., *71*, 1942, 253–259; Wharton and Wharton, Am. Jour. Hyg., *19*, 1934, 189–216.

7. **Tarpeia vulpis** *spec. nov.* From Latin *vulpes*, fox.

Common name: Fox-encephalitis virus.

Hosts: *CANIDAE—Vulpes* sp., silver fox. Experimentally, also some, but not all, dogs; coyote.

Insusceptible species: Gray fox, mink, ferret, sheep, laboratory rabbit.

Geographical distribution: United States.

Induced disease: In fox, after 2 days from time of infection, loss of appetite, slight nasal discharge; convulsions with early death or hyperexcitability, blind walking, lethargy, flaccid or spastic paralysis, muscular twitching, fearfulness, weakness, coma and death; many more foxes become infected in epizootics than show obvious disease, some being symptomless carriers; 12 to 20 per cent fatalities may be experienced among young foxes on ranches, 3 to 9 per cent among adults. Intranuclear inclusions in vascular endothelial cells especially in cerebral endothelium; sometimes in hepatic cells and endothelial cells of liver and kidney; no intracytoplasmic inclusions; virus in heart blood, spleen, and brain; in carriers, virus is believed to persist in focal lesions in upper respiratory tract. Experimentally in susceptible dogs, sometimes coryza, discharge from eyes and nose often purulent, commonly fits of excitement, coma, death; recovery rare; cellular infiltration in the central nervous system, focal necrosis of the liver; specific intranuclear inclusions in cells of the vascular endothelium, meningeal cells, reticulo-endothelium, hepatic cells, and occasionally in cortical cells of the adrenal.

Transmission: Experimentally, by skin scarification, intramuscular injection, intraperitoneal injection, inoculation of cisterna, intratesticular injection, inoculation of nasal cavity; not by corneal scarification.

Immunological relationships: Injections of this virus afford no immunity to subsequent infection by canine distemper virus.

Filterability: Passes Berkefeld N filter.

Other properties: Viable in 50 per cent glycerine for several years, in carcass for several days.

Literature: Barton and Green, Am. Jour. Hyg., *37*, 1943, 21–36; Green, Proc. Soc. Exp. Biol. and Med., *23*, 1926, 677–678; Am. Jour. Hyg., *18*, 1931, 201–223; Green and Dewey, Proc. Soc. Exp. Biol. and Med., *27*, 1929, 129–130; Green and Evans, Am. Jour. Hyg., *29* (B), 1939, 73–87; Green and Shillinger, *ibid.*, *19*, 1934, 362–391; Green et al., *ibid.*, *12*, 1930, 109–129; *14*, 1931, 353–373; *18*, 1933, 462–481; *19*, 1934, 343–361; *21*, 1935, 366–388; *24*, 1936, 57–70; Lucas, Am. Jour. Path., *16*, 1940, 739–760.

8. **Tarpeia viverrae** *spec. nov.* From Latin *viverra*, ferret.

Common name: Ferret-distemper virus.

Host: *MUSTELIDAE—Mustela furo*, ferret.

Insusceptible species: Dog, mouse, rat, guinea pig, rabbit.

Geographical distribution: United States.

Induced disease: In ferret, fever to 105 or 106° F, lethargy, loss of appetite, conjunctivitis with exudate closing eyes, sometimes a purulant nasal discharge, weight loss small, sneezing rare, difficulty in breathing, death 14 to 56 days after inoculation (average 20 days), sometimes

preceded by convulsions and other nervous signs; fatality rate 70 to 100 per cent.

Transmission: By cage contacts. By feeding. Experimentally by intranasal, subcutaneous, or intradermal inoculation.

Immunological relationships: In immunized animals, no cross immunity with canine distemper virus nor with human influenza virus.

Thermal inactivation: At 60° C in 30 minutes.

Filterability: Passes Berkefeld N filter.

Other properties: Viable at least 3, but not 5, months in 50 per cent neutral glycerine; at least 4 months when frozen and dried *in vacuo*.

Literature: Slanetz and Smetana, Jour. Exp. Med., *66*, 1937, 653–666; Spooner, Jour. Hyg., *38*, 1938, 79–89.

9. **Tarpeia avium** *spec. nov.* From Latin *aves*, fowl of the air.

Common names: Laryngotracheitis virus; also known as infectious laryngotracheitis virus and as infectious bronchitis virus.

Hosts: *PHASIANIDAE—Gallus gallus* (L.), chicken. Experimentally, also *PHASIANIDAE*—pheasant; F_1 hybrid between male Ringneck pheasant and female bantam chicken; chorioallantoic membrane of developing chicken embryo (with macroscopic lesions on membrane as a result of proliferative and necrotic changes); turkey embryo.

Insusceptible species: Guinea fowl (no evidence of disease on inoculation); white rat, guinea pig, rabbit; embryos of pigeon, guinea fowl, and duck.

Geographical distribution: United States, Canada, Australia.

Induced disease: In domestic chicken, mostly among pullets and yearling hens, loss of appetite, lachrymation from one or both eyes, respiratory distress, hemorrhagic and mucous exudate in lumen of trachea and occasionally in the bronchi; death as a result of asphyxiation or, more often, recovery; recovered birds occasionally carry the virus in the upper respiratory tract for some time (a period of 467 days has been recorded); virus is not found on eggs during an outbreak in a flock, but is always in trachea of an affected bird; intranuclear inclusions in tracheal lesions; virus has special affinity for mucous membrane of eye, nostril, larynx, trachea, cloaca, and bursa of Fabricius; usually affects more than half the birds in a flock, with a mortality of 5 to 60 per cent (averaging between 10 and 20 per cent).

Transmission: By contacts. Experimentally, by intrabursal injection (in bursa of Fabricius) or by rubbing the mucous membrane in the dorsal region of the outer or proctodeal part of the cloaca with a small cotton swab moistened with a suspension of virus.

Serological relationships: Serum from recovered fowl neutralizes virus; dilution tends to reactivate neutralized virus.

Immunological relationships: Experimental infection of cloaca and bursa of Fabricius, especially in 2 to 4-month-old birds, immunizes against infection by subsequent tracheal inoculation.

Thermal inactivation: At 55.5° C in 10 to 15 minutes; at 60° C in 2 to 3 minutes; at 75° C in ¼ to ½ minute; all tests with virus in the presence of tracheal exudate.

Filterability: Passes Berkefeld V and N filters.

Strains: A Victorian strain has been reported as of low virulence for fowls.

Other properties: Inactivated in 5 per cent phenol in 1 minute; in 3 per cent cresol compound in ½ minute; in 1 per cent sodium hydroxide in ½ minute. Viable in tracheal fluid in dark for 75, not 110, days; in light for 6, not 7, hours; in buffer solution at pH 7.4 for 131 days; at 4 to 10° C in dark for at least 217 days; in dried state for at least 661 days. Viable in dead body at 37° C for 22, not 44, hours; at 13 to 23° C for 10, not 15, days; at 4 to 10° C for 30, not 60, days.

Literature: Beach, Science, *78*, 1930, 633–634; Jour. Exp. Med., *54*, 1931, 809–816; Jour. Inf. Dis., *57*, 1935, 133–135;

Beach et al., Poultry Science, *13*, 1934, 218–226; Beaudette and Hudson, Science, *76*, 1932, 34; Jour. Am. Vet. Med. Assoc., *82* (N.S. *35*), 1933, 460–476; *95*, 1939, 333–339; Brandly, *ibid.*, *88* (N.S. *41*), 1936, 587–599; Jour. Inf. Dis., *57*, 1935, 201–206; Brandly and Bushnell, Poultry Science, *13*, 1934, 212–217; Burnet, Brit. Jour. Exp. Path., *15*, 1934, 52–55; Jour. Exp. Med., *63*, 1936, 685–701; Burnet and Foley, Austral. Jour. Exp. Biol. and Med. Sci., *19*, 1941, 235–240; Gibbs, Jour. Am. Vet. Med. Assoc., *81*, (N.S. *34*), 1932, 651–654; Massachusetts Agr. Exp. Sta., Bull. 295, 1933, *ibid.*, Bull. *311*, 1934; Hinshaw et al., Poultry Science, *10*, 1931, 375–382; Hudson and Beaudette, Science, 76, 1932, 34; Cornell Vet., *22*, 1932, 70–74; Kernohan, California Agr. Exp. Sta., Bull. 494, 1930, 3–22; Jour. Am. Vet. Med. Assoc., *78* (N.S. *31*), 1931, 553–555; Komarov and Beaudette, Poultry Science, *11*, 1932, 335–338; May and Tittsler, Jour. Am. Vet. Med. Assoc., *67*, (N.S. *20*), 1925, 229–231; Schalm and Beach, Jour. Inf. Dis., *56*, 1935, 210–223; Seifried, Jour. Exp. Med., *54*, 1931, 817–826.

Genus III. Tortor *gen. nov.*

Viruses of the Hog-Cholera Group, inducing diseases characterized by involvement of many tissues. Generic name from Latin *tortor*, tormentor.

The type species is *Tortor suis spec. nov.*

Key to the species of genus Tortor.

I. In mammals.
  A. Infecting swine. — 1. *Tortor suis.*
  B. Infecting cattle. — 2. *Tortor bovis.*
  C. Infecting the horse. — 3. *Tortor equorum.* 4. *Tortor equae.*
  D. Infecting sheep. — 5. *Tortor ovis.*
  E. Infecting cat. — 6. *Tortor felis.*
II. In birds. — 7. *Tortor galli.* 8. *Tortor furens.*

1. **Tortor suis** *spec. nov.* From Latin *sus*, hog.

Common names: Hog-cholera virus, swine-fever virus.

Host: *SUIDAE—Sus scrofa* L., domestic swine. Warthog (symptomless carrier).

Insusceptible species: Dog, cat, cow, horse, donkey, sheep, goat, rabbit, guinea pig, mouse, rat, goose, hen, duck, pigeon.

Geographical distribution: Almost universal in pig-breeding countries, especially in Europe, the British Isles, North and South America.

Induced disease: In swine, after intramuscular injection, increased temperature and prostration within 2½ to 3 days; later lymph nodes enlarged, sometimes hemorrhagic; hemorrhages under capsule of kidneys. Virus may remain in blood of recovered pigs for 10 months. Acquired immunity is lasting, but most naturally infected animals die in newly infected herds. Virus has been cultured

in minced swine testicle on solid serum-agar and on egg membrane, increase being limited to the living tissues from the swine and furnishing inoculum active in amounts as small as 10^{-5} ml.

Transmission: By feeding. Through air contamination. Rarely by contact. Experimentally, by subcutaneous injection. Urine highly infective. Virus in blood and all tissues early in disease.

Serological relationships: Immune serum affords passive protection.

Thermal inactivation: At 55° C in 30 minutes; at 60° C in 10 minutes. At 72° C in 1 hour in dried blood.

Filterability: Passes Berkefeld filter.

Other properties: Viable in blood in cool, dark place at least 6 years.

Literature: De Kock et al., Onderstepoort Jour. Vet. Sci. and Anim. Indust., *14*, 1940, 31–93; Hecke, Cent. f. Bakt., I Abt., Orig., *126*, 1932, 517–526; Montgomery, Jour. Comp. Path. and Therap., *34*, 1921, 159–191; Röhrer, Arch. Tierheilk., *62*, 1930, 345–372, 439–462; *64*, 1931, 124–143; TenBroeck, Jour. Exp. Med., *74*, 1941, 427–432.

2. **Tortor bovis** *spec. nov.* From Latin *bos*, cow.

Common names: Cattle-plague virus, virus of pestis bovina, runderpest virus, Rinderpest virus.

Hosts: *BOVIDAE—Bos taurus* L., domestic cattle; swine, buffalo, zebu cattle, sheep, goat, camel, deer. Koedoe, eland, bushbuck, duiker, and other antelopes.

Insusceptible species: Man, solipeds, carnivora.

Geographical distribution: Widespread over Asia and the Asiatic islands. At times in Western Europe. Enzootically in Turkey. Periodically in North Africa, especially in Egypt; at times throughout Africa. Not in North America. At times in South America, Australia (suppressed quickly).

Induced disease: In domestic cattle, after 3 to 9 days, febrile reaction, restlessness, loss of appetite, cessation of rumination; fever highest at 5th or 6th day of disease, then temperature drops to normal or subnormal and diarrhea begins; muzzle dry, coat staring, hair dull, skin moist in parts; twitching of superficial muscles, grinding of teeth, arching of back, glairy discharge from nose, redness of mucous membranes; restlessness increases, diarrhea becomes severe with fetid, blood-stained or blackish liquid discharges; weakness, drooping of ears, occasional yawning, coldness of extremities; occasionally excitement precedes weakness; skin may become red and moist, showing protuberances and vesicles, with matted hair; later wrinkling and scab formation; conjunctiva red, eyelids swollen, tears flowing, followed by mucous, then purulent, discharge; sometimes a cough develops and respirations become rapid; red spots inside mouth develop into erosions or ulcers, often confluent; pregnant animals often abort; milk of cows decreases, sometimes becoming yellow and watery. Death is sometimes early (1 to 2 days after first manifestations of disease), more often delayed (4 to 7 days); sometimes animals live 2 or 3 weeks or longer. Disease milder and more chronic where enzootic; morbidity to 100 per cent and mortality to 96 per cent in new areas. Recovered animals show a lasting, sterile mmunity. Urine, feces, nasal and lachrymal discharges, sweat, aqueous humour, cerebrospinal fluid, lymph, emulsions of viscera and muscles, and blood are infective during the course of the disease.

Transmission: By contact, even during prodromal period; by contaminated food, troughs, or other articles. No insect vector is known.

Immunological relationships: One attack confers a lasting immunity, except rarely, when a mild second attack may occur. A calf from a diseased mother may be resistant if pregnancy was far advanced when the disease occurred.

Filterability: Passes Berkefeld V filter candle, with difficulty.

Other properties: Remains infective at

least 2 weeks at 0° C in virulent blood, less than 2 days in hides dried in direct sunlight, 3 days in contaminated wool, as long as 12 days in meat; is inactivated by glycerine, bile, chloroform, formalin, and 2 per cent phenol; is virulent at least 25 days in body of leech, *Hirudo boyntoni* Wharton (*HIRUDIDAE*), fed on sick animal.

Literature: Boynton, Philippine Agr. Rev., *10*, 1917, 410–433; Daubney, Jour. Comp. Path. and Therap., *41*, 1928, 228–248; 263–297; Hornby, *ibid.*, *41*, 1928, 17–24; Pfaff, Onderstepoort Jour. Vet. Sci. and Anim. Indust., *11*, 1938, 263–330; *15*, 1940, 175–184; Weston, Jour. Am. Vet. Med. Assoc., *66* (N.S. *19*), 1924, 337–350.

3. **Tortor equorum** *spec. nov.* From Latin *equus*, horse.

Common names: Horse-sickness virus, African horse-sickness virus, virus of pestis equorum, virus of perdesiekte, virus of South African Pferdesterbe.

Hosts: *EQUIDAE—Equus caballus* L., horse; perhaps *E. asinus* L., donkey. Experimentally, also *CANIDAE—Canis familiaris* L., dog. *CAVIIDAE—Cavia porcellus* (L.), guinea pig. *MURIDAE —Rattus norwegicus* (Erxleben), wild and albino rat; mouse; Angora goat; *Mastomys coucha*, multimammate mouse; *Tatera lobengula*, gerbille; chick embryo (but no virus in hatched chick). Mule and zebra relatively resistant.

Insusceptible species: *HOMINIDAE —Homo sapiens* L., man. *LEPORIDAE—Oryctolagus cuniculus* (L.), rabbit (no observed disease).

Geographical distribution: Africa, especially in coastal regions and river valleys.

Induced disease: In the horse, four types of disease are recognized. Horse-sickness fever, prodromal period 5 to 28 days, rise of body temperature to 105° F in 1 to 3 days, with return to normal temperatures in another day or two; sometimes loss of appetite, redness of conjunctiva, labored breathing, and accelerated pulse; recovery prompt. Dunkop or acute pulmonary horse-sickness, prodromal period of 3 to 5 days, severe dyspnea, fever, coughing, frothing at nostrils; fever to 106° F, breathing rate to 60 a minute, nostrils dilated, head and neck extended, ears drooping, sweating, progressive weakness; often fatal. Dikkop, or cardiac form of horse-sickness, prodromal period 5 to 21 days, fever develops slowly, lasts long; edematous swellings of head and neck, symptoms of cardiac dyspnea, sometimes blood spots on conjunctiva, mucous membranes of mouth and tongue bluish, restlessness; sometimes fatal outcome. Mixed form of horse-sickness, combining features of pulmonary and cardiac types. Horses recovering from natural infections are known as "salted" and possess heightened resistance to the disease.

Transmission: Not by contact. Mosquitoes and biting flies have been suspected as vectors. Experimentally, by intravenous or subcutaneous injection.

Serological relationships: Serologically distinguishable strains exist.

Immunological relationships: Immunity to homologous strain complete after an attack (horse then known as "salted" for that strain), but immunity to heterologous strains incomplete. Antibodies absent from young at birth but as high in titer as in dam within 30 hours, presumably from colostral milk; declining gradually over a period of about 6 months.

Thermal inactivation: At 57.5 to 60° C in 10 minutes.

Filterability: Passes Berkefeld, Chamberland F, and Seitz EK filters.

Other properties: Viable dry at least 15 months. Stable in alkaline solutions (to pH 10), unstable in acid (beyond pH 6.0). Serum-saline solutions preferable to saline solutions for storage. Particle diameter determined as 40 to 60 millimicrons (mean 50 millimicrons) by filtration methods, 45.4 millimicrons by centrifuging. Density 1.25 gm per ml. Isoelectric point at pH 4.8.

Literature: Alexander, Onderstepoort Jour. Vet. Sci. and Anim. Indust., *4*, 1935, 291–322, 323–348, 349–377, 379–388; *7*, 1936, 11–16; *11*, 1938, 9–19; Alexander and DuToit, *ibid.*, *2*, 1934, 375–391; Alexander and Mason, *ibid.*, *16*, 1941, 19–32; Alexander et al., *ibid.*, *7*, 1936, 17–30; DuToit et al., *ibid.*, *1*, 1933, 21–24, 25–50; Henning, in Animal Diseases in South Africa, Central News Agency, Limited, South Africa, *2*, 1932, 516–538; M'Fadyean, Jour. Comp. Path. and Therap., *13*, 1900, 1–20; *23*, 1910, 27–33, 325–328; Nieschulz and DuToit, Onderstepoort Jour. Vet. Med. and Anim. Indust., *8*, 1937, 213–268; Polson, *ibid.*, *16*, 1941, 33–50, 51–66; Nature, *148*, 1941, 593–594; Theiler, Deutsch. tierärztl. Wochenschr., *9*, 1901, 201–203, 221–226, 233–237, 241–242; Report for 1905–1906 of the Govt. Veterinary Bacteriologist, Transvaal Dept. Agr., 1907, 160–162; Jour. Comp. Path. and Therap., *23*, 1910, 315–325.

4. **Tortor equae** *spec. nov.* From Latin *equa*, mare.

Common name: Mare-abortion virus.

Hosts: *EQUIDAE—Equus caballus* L., horse. Experimentally, also Syrian hamster (newborn); tissues of human placenta grafted on the chorioallantois of the chick embryo.

Insusceptible species: Chicken (embryo; no observed susceptibility).

Induced disease: In horse, small, multiple, grayish white areas of necrosis in the livers of aborted fetuses; acidophilic intranuclear inclusions in hepatic cells around these foci, in epithelial cells of bile ducts, and in bronchial epithelium; petechial hemorrhages in the heart, spleen, and lungs; excess fluid in the thoracic cavity.

Transmission: By contact. By living in contaminated stalls.

Literature: Anderson and Goodpasture, Am. Jour. Path., *18*, 1942, 555–561; Dimock, Jour. Am. Vet. Med. Assoc., *96*, 1940, 665–666; Dimock and Edwards, Cornell Vet., *26*, 1936, 231–240; Goodpasture and Anderson, Am. Jour. Path., *18*, 1942, 563–575; Hupbauer, Münch. Tierärztl. Wchnschr., *89*, 1938, 37–38; Miessner and Harms, Deutsche Tierärztl. Wchnschr., *46*, 1938, 745–748.

5. **Tortor ovis** *spec. nov.* From Latin *ovis*, sheep.

Common name: Blue-tongue virus.

Hosts: *BOVIDAE—Ovis aries* L., sheep; *Bos taurus* L., cattle.

Geographical distribution: South Africa.

Induced disease: Both sheep and cattle may carry the virus at times without obvious manifestations of disease or there may be severe manifestations. In sheep, experimentally, diffuse hyperemia of buccal mucosa, especially of lips; then petechiae and ecchymoses followed by excoriations and necrosis of the mucous membrane, especially on lips, tongue, inside of cheeks, dental pad, gums, muzzle, and external nares; sometimes deep seated necrotic ulcers on tongue developing from the more usual superficial necrotic process; mucoid discharge from nostrils, becoming muco-hemorrhagic; commonly frothing at the mouth in early stages of the disease; frequently reddening of skin of lips and nose; rarely whole skin becomes flushed and wool is shed; often swelling of vulva with necrotic changes on borders and petechiae in mucosa; tongue sometimes swollen; lameness common and severe; recovery or death. In cattle, edema of lips and tongue; hyperemia of oral mucosa; multiple hemorrhages in skin, lips, mucous membrane of the lips, tongue, dental pad, buccal cavity, small intestine, myocardium, epicardium, and endocardium, less frequently in the trachea, nasal cavity, bladder, urethra, pulmonary artery, and pleura; localized necrotic areas followed by ulceration on lips, gums, the dental pad, tongue, mucous membrane of the rumen, pylorus of the stomach, and the external nares; scattered skin lesions with reddening, slight exudation, crusting, sloughing of crusts and hair together,

mucoid or mucopurulent discharge from nostrils; prognosis favorable in mild cases, but disease occasionally terminates with death.

Transmission: Not by contact; arthropod vector suspected.

Other properties: Infective particle calculated to be 87 to 105 millimicrons in diameter by sedimentation studies, 100 to 132 millimicrons in diameter by ultrafiltration.

Literature: Bekker et al., Onderstepoort Jour. Vet. Sci. and Anim. Indust., *2*, 1934, 393–507; De Kock et al., *ibid.*, *8*, 1937, 129–180; Henning, in Henning, M. W., Animal Diseases in South Africa, Central News Agency, Ltd., South Africa, 1932, vol. *2*, chapter 27, pages 503–515; Mason and Neitz, Onderstepoort Jour. Vet. Sci. and Anim. Indust., *15*, 1940, 149–157; Nieschulz et al., *ibid.*, *2*, 1934, 509–562; Polson, Nature, *148*, 1941, 593–594.

6. **Tortor felis** *spec. nov.* From Latin *feles*, cat.

Common names: Panleucopenia virus, infectious feline agranulocytosis virus, infectious aleucocytosis virus, feline enteritis virus.

Host: *FELIDAE—Felis catus* L., domestic cat.

Insusceptible species: White mouse, guinea pig, domestic rabbit, ferret; *Citellus richardsonii* (Sabine), ground squirrel.

Geographical distribution: United States, Germany.

Induced disease: In cat, variable effects, some individuals little affected, others listless, recumbent, refusing food, showing some vomiting, diarrhea, nasal and ocular discharges; often death, after a few minutes of fibrillary twitching and terminal clonic convulsions, before there is much loss of weight; sometimes recovery with return of appetite. Profound leucopenia and marked relative lymphocytosis without thrombopenia or appreciable anemia; proliferation of reticuloendothelial cells of lymph nodes and spleen; intranuclear inclusion in cells of gastro-intestinal mucosa, spleen, lymph nodes, bone marrow, and bronchial mucosa.

Transmission: Perhaps by nasal droplets or contaminated food. No arthropod vector recognized. Experimentally by oral, intragastric, cutaneous, subcutaneous, intraperitoneal, intravenous, and intranasal routes.

Serological relationships: Sera from panleucopenia-immune cats protects against agranulocytosis virus.

Immunological relationships: Cats immune as a result of earlier infection with agranulocytosis virus resist later inoculation with panleucopenia virus. Previous inoculation ineffective if made with hog-cholera virus or fox-encephalitis virus.

Filterability: Passes Berkefeld V, N, and W filters and Seitz EK discs.

Other properties: Remains active in 50 per cent glycerine at least 138 days in tissues; not inactivated by drying while frozen, nor by freezing at about −80° C.

Literature: Hammon and Enders, Jour. Exp. Med., *69*, 1939, 327–352; *70*, 1939, 557–564; Kikuth et al., Cent. f. Bakt., I Abt., Orig., *146*, 1940, 1–17; Lawrence and Syverton, Proc. Soc. Exp. Biol. and Med., *38*, 1938, 914–918; Lawrence et al., Jour. Exp. Med., *77*, 1943, 57–64; Am. Jour. Path., *16*, 1940, 333–354; Syverton et al., Jour. Exp. Med., *77*, 1943, 41–56.

7. **Tortor galli** *spec. nov.* From Latin *gallus*, cock.

Common names: Fowl-plague virus, fowl-pest virus.

Hosts: Chiefly chicken, turkey, goose. Experimentally, also ferret, rhesus monkey, hedgehog, pigeon, duck, canary, mouse, rat, rabbit. Multiplies in embryonated hen's egg; edema, but no discrete primary lesions in chorioallantoic membrane.

Geographical distribution: Widespread throughout Europe, North and South America, Asia.

Induced disease: In chicken, loss of appetite, tendency to leave companions

and seek shade, drooping of wings and tail; eyes closed or partly closed; some dyspnea; in some cases, edema of head and neck; in late stages, sometimes cyanosis of comb and skin; staggering, twitching, or spasms; fever may disappear and temperature become subnormal before death; recovery in about 30 per cent of all cases; linear and punctiform hemorrhages throughout body.

Transmission: Method of natural transmission unknown. The fowl louse, *Goniocides dissimilis* (*PHILOPTERIDAE*), has been suspected as vector (Maggiora and Tombolato, Rendiconti, Accademia delle Scienze dell'Instituto di Bologna, n.s. *27*, 1923, 200–203). Experimentally, by subcutaneous, intramuscular, and intravenous injection.

Serological relationships: Specific neutralizing antiserum does not react with influenza virus. No reaction of fowl-plague virus with antisera specific for canine distemper, influenza, or Rift Valley fever viruses.

Thermal inactivation: At 55° C in 1 hour in whole blood or brain.

Filterability: Passes membrane of average pore diameter 150, not 100, not ordinarily 125, millimicrons. Passes Berkefeld and Chamberland filters.

Other properties: Particle diameter estimated by filtration as 60 to 90 millimicrons; by centrifugation, as 120 to 130 millimicrons. Viable after exposure in 1:10,000 dilution for 10 minutes, in 2 mm layer of 1:50,000 methylene blue, 15 cm from a 300 candle-power filament lamp. Withstands drying. Precipitates from salt-free solutions or in presence of half-saturated ammonium sulphate solutions; virus held to be of globulin nature by Mrowka, Cent. f. Bakt., I Abt., Orig., *67*, 1912, 249–268.

Strains: Variant strains have been produced by intracerebral passage in brains of canaries and mice.

Literature: Bechhold and Schlesinger, Biochem. Ztschr., *236*, 1931, 387–414; Ztschr. Hyg. Infektionskr., *112*, 1931, 668–679; Burnet and Ferry, Brit. Jour. Exp. Path., *15*, 1934, 56–64; Centanni, Cent. f. Bakt., I Abt., Orig., *51*, 1902, 145–152, 182–201; Elford and Todd, Brit. Jour. Exp. Path., *14*, 1933, 240–246; Findlay and Mackenzie, *ibid.*, *18*, 1937, 146–155, 258–264; Findlay et al., Jour. Path. and Bact., *45*, 1937, 589–596; Lépine Compt. rend. Soc. Biol., Paris, *121*, 1936, 509–510; Mackenzie and Findlay, Brit. Jour. Exp. Path., *18*, 1937, 138–145; Nieschulz and Bos, Cent. f. Bakt., I Abt., Orig., *131*, 1934, 1–6; Plotz and Haber, Compt. rend. Soc. Biol., Paris, *125*, 1937, 339–340.

8. **Tortor furens** *spec. nov.* From Latin *furere*, to rage.

Common name: Newcastle-disease virus.

Hosts: *PHASIANIDAE—Gallus gallus* (L.), domestic chicken. *HOMINIDAE—Homo sapiens* L., man (by laboratory accident). Experimentally, also pigeon; chick embryo (with primary lesions and cytoplasmic inclusions in chorioallantoic membrane).

Geographical distribution: England, probably also East Indies, Korea, Japan, India, Australia.

Induced disease: In chicken, acute, febrile, highly contagious, usually fatal disease resembling fowl plague; loss of appetite, crouching attitude, half closed eyes, rapid respirations, watery yellowish-white diarrhea with nauseating odor; death usually between 6th and 8th day. In man, accidentally infected in laboratory by virus sprayed into eye, virus recoverable from temporarily inflamed eye; recovery in 8 days with gradual increase of specific antibodies in blood.

Transmission: By contact between healthy and diseased birds.

Serological relationships: Antiserum effective in neutralizing homologous virus.

Immunological relationships: Chickens immune to infection by fowl-plague virus are susceptible to infection by this virus

and *vice versa*. Immunization to this virus does not decrease susceptibility to comb or mouth form of fowl pox.

Thermal inactivation: At 60° C in 1 hour; not at 56° C in 30 minutes.

Filterability: Passes Berkefeld, Chamberland L_3, and Seitz filters.

Other properties: Particle diameter calculated from filtration experiments to be 80 to 120 millimicrons. Not inactivated in 30 minutes in 1:50,000 methylene blue solution in 2 mm layer 15 cm from a 300 candle-power filament lamp.

Literature: Burnet, Med. Jour. Australia, *30*, 1943, 313–314; Burnet and Ferry, Brit. Jour. Exp. Path., *15*, 1934, 56–64; Doyle, Jour. Comp. Path. and Therap., *40*, 1927, 144–169.

FAMILY V. TRIFURACEAE *FAM. NOV.*

Viruses of the Infectious Anemia Group, inducing diseases mainly characterized by disturbances in balance of blood cells. There is a single genus.

Genus Trifur *gen. nov.*

With characters of the family. Generic name from Latin *trifur*, arrant thief. The type species is *Trifur equorum spec. nov.*

Key to the species of genus Trifur.

I. Affecting horse. 1. *Trifur equorum.*

II. Affecting fowl. 2. *Trifur gallinarum.*

1. **Trifur equorum** *spec. nov.* From Latin *equus*, horse.

Common name: Equine infectious-anemia virus.

Hosts: *EQUIDAE—Equus caballus* L., horse; *E. asinus* L., donkey. *HOMINIDAE—Homo sapiens* L., man. Experimentally, also *EQUIDAE—Equus asinus* × *E. caballus*, mule. *SUIDAE—Sus scrofa* L., swine.

Insusceptible species: *BOVIDAE—Bos taurus* L., cattle; *Ovis aries* L., sheep; *Capra hircus* L., goat. *CANIDAE—Canis familiaris* L., dog.

Geographical distribution: Europe, Union of South Africa, United States, Canada, Japan; at times in most parts of the world; not Australia.

Induced disease: In horse, progressive anemia with eventual death or clinical recovery and retention of virus; disease may be acute, subacute, or chronic; in acute disease, temperature rise to 104 to 105° F. or even 106 to 107° F, remaining high much of the time until death or change to subacute or chronic form; in the acute form of the disease there is dullness, decreased appetite, drooping of head, flexing of limb not supporting weight; sometimes increase in pulse frequency to 70 or even 100 a minute but oftener rates around 50 a minute; conjunctiva sometimes colored orange, with injection of vessels and petechiae, later becoming muddy colored or pale red, membrane edematous; uncertain gait, trailing of hind feet, prostration, sometimes death; subacute disease milder and with remissions; chronic disease still milder, anemia conspicuous, sometimes death from debility or at end of a febrile attack; blood infective long (3 to 7 years) after clinical recovery; urine infective to horse by mouth. In man, diarrhea alternating with constipation, herpes-like exanthema on abdominal wall, blood sometimes in feces; persistent headache, temperature normal; later, lumbar pains, generalized edema, general debility, loss of flesh, pallor of face and mucosae; filtered blood in 1 ml. amount fatal to horse, inducing infectious anemia; improvement after 2 to 4 years. In swine, experimentally, sometimes no outward and obvious signs of disease but blood abnormal and infective; sometimes severe anemia, fever, prostration, loss of appetite.

Thermal inactivation: At 58 to 60° C in 1 hour.

Filterability: Passes Berkefeld V filter candle.

Other properties: Viable in blood in citrate saline at −2° C for at least a year. Drying does not inactivate in 10 days but does in 1 month.

Literature: DeKock, Union of South Africa, Dept. of Agr., 9th and 10th Reports for 1923, Pretoria 1924, 253-313; Habersang, Monatshefte für prakt. Tierheilk., *30*, 1920, 171-176; Kutsche, *ibid.*, *30*, 1920, 557-568; Peters, Jour. Am. Vet.

Med. Assoc., *66*, 1924, 363–366; Theiler and Kehoe, Union of South Africa, Dept. of Agr., 3rd and 4th Reports of the Director of Veterinary Research, 1915, 215–289.

2. **Trifur gallinarum** *spec. nov.* From Latin *gallina*, hen.

Common name: Fowl-leucosis virus.

Host: *Gallus gallus* (L.), chicken.

Geographical distribution: United States, England, Europe.

Induced disease: In chicken, neurolymphomatosis, with eye lesions (slate gray or bluish color replacing normal bay color of iris), anemia, hemocytoblastosis, lymphoid, erythroid or myeloid types of leucosis; the hemocytoblastosis is followed by infiltration of the central nervous system, peripheral nerves, iris, and many visceral organs by hemocytoblasts and lymphocytes, producing lesions sometimes resembling neoplasms and consisting chiefly of hemocytoblasts (hemocytoblastomata); marrow of radius and ulna becomes hyperplastic; virus in blood plasma, blood cells, emulsions of organs; blood normal in its hydrogen-ion concentration; recovery never complete; some stocks less susceptible than others.

Transmission: By pen contact or contaminated litter. Experimentally by intravenous injection of cell-free filtrates. Not by the mosquitoes, *Culex pipiens* and *Aëdes aegypti* (*CULICIDAE*). Day-old chicks from iritis parents contain the infective agent and show some form of the induced disease in 80 per cent of the progeny if both parents show iritis, in 70 per cent if male is normal, 15 per cent if female is normal.

Serological relationships: Specific neutralizing antibodies are formed in the rabbit as a result of injecting infective materials partly purified by sedimentation in the ultracentrifuge.

Thermal inactivation: At 56° C in 30 minutes.

Filterability: Passes Berkefeld V, N, and W filter candles; 1.5 per cent, but not often 3 per cent, collodion membranes; Seitz asbestos filter.

Other properties: Viable after drying at least 54 days, in glycerine at least 104 days, at 4° C at least 14 days, at −60° C at least 6 months; after freezing and thawing, and after freezing in liquid air. Not viable after 14 days at 37.5° C. Particle diameter between 100 and 400 millimicrons.

Literature: Ellermann and Bang, Cent. f. Bakt., I Abt., Orig., *46*, 1908, 4–5, 595–609; Furth, Proc. Soc. Exp. Biol. and Med., *27*, 1929, 155–157; Jour. Exp. Med., *53*, 1931, 243–267; *55*, 1932, 465–478, 495–504; *58*, 1933, 253–275; *59*, 1934, 501–517; Furth and Miller, *ibid.*, *55*, 1932, 479–493; Hall et al., Am. Jour. Vet. Res., *2*, 1941, 272–279; Jármai, Arch. wissensch. u. prakt. Tierhielk., *62*, 1930, 113–131; Johnson, Virginia Agr. Exp. Sta. Tech. Bull. *56*, 1934, 1–32; Johnson and Bell, Jour. Inf. Dis., *58*, 1936, 342–348; Kabat and Furth, Jour. Exp. Med., *71*, 1940, 55–70; *74*, 1941, 257–261; Lee and Wilcke, Am. Jour. Vet. Res., *2*, 1941, 292–294; Lee et al., Jour. Infect. Dis., *61*, 1937, 1–20; Pierce, Am. Jour. Path., *18*, 1942, 1127–1139; Ratcliffe and Stubbs, Jour. Inf. Dis., *56*, 1935, 301–304.

FAMILY VI. RABULACEAE *FAM. NOV.*

Viruses of the Mumps Group, characterized in general by a special affinity for tissues of the salivary glands. There is a single genus,

Genus I. Rabula *gen. nov.*

With characters of the family. Generic name from Latin *rabula*, pettifogger. The type species is *Rabula inflans spec. nov.*

Key to species of the genus **Rabula.**

I. Affecting man.	1. *Rabula inflans.*
II. Affecting guinea pig.	2. *Rabula levis.*
III. Affecting hamster.	3. *Rabula innocuus.*
IV. Affecting rat.	4. *Rabula exiguus.*
V. Affecting mouse.	5. *Rabula latens.*

1. Rabula inflans *spec. nov.* From Latin *inflare*, to puff up.

Common names: Mumps virus, virus of epidemic parotitis.

Hosts: *HOMINIDAE—Homo sapiens* L., man. Experimentally, also *CERCOPITHECIDAE—Macaca mulatta* (Zimmermann), rhesus monkey. *FELIDAE —Felis catus* L., domestic cat.

Geographical distribution: World-wide.

Induced disease: In man, in order of frequency, parotitis, orchitis, meningoencephalitis, pancreatitis, or ovaritis; rarely fatal; when parotitis occurs, onset is sudden, with pain in one or both parotid glands, sometimes also with involvement of submaxillary and sublingual glands, swelling and malaise gradually disappearing within a week or 10 days; there is virus in saliva 48 hours after onset; orchitis, less common, is usually unilateral and may be accompanied by some epididymitis. In rhesus monkey, experimentally, acute, non-suppurative parotitis; focal necrosis in acinar epithelial cells of parotid gland, and secondary inflammation; dissemination of lesions within the gland, enlargement of gland to palpation and pitting edema of jowl 6 to 8 days after inoculation, often with a rise of temperature; cytoplasmic inclusion bodies affected glands, staining pink, round or oval, 3 to 10 microns in diameter, often vacuolate, usually surrounded by a narrow clear zone in the cytoplasm; blood and uninoculated salivary gland of affected animal not effective sources of virus.

Transmission: Probably by droplets arising directly from infected individuals. Experimentally, by injecting sterile fluids containing virus into Stenson's duct of parotid gland in *Macaca mulatta.*

Serological relationships: A specific complement-fixing antibody occurs in human and monkey convalescent serum and is demonstrable by the use of monkey-gland antigen.

Immunological relationships: Specific immunity induced by attack; passive immunization rarely successful.

Thermal inactivation: At 55° C in 1 hour.

Filterability: Passes Berkefeld V and N filter candles.

Other properties: Viable in 50 per cent glycerine at 2° C at least 5 weeks, in 50 per cent glycerine at 10° C. at least 7 weeks, dried while frozen at least 7 weeks, in frozen saliva at least 3 weeks.

Literature: Bloch, Am. Jour. Path., *13*, 1937, 939–944; Enders and Cohen, Proc. Soc. Exp. Biol. and Med., *50*, 1942, 180–184; Findlay and Clarke, Brit. Jour. Exp. Path., *15*, 1934, 309–313; Johnson and Goodpasture, Jour. Exp. Med., *59*, 1934, 1–19; Am. Jour. Hyg., *21*, 1935, 46–57; *23*, 1936, 329–339; Am. Jour. Path., *12*, 1936, 495–510.

2. **Rabula levis** *spec. nov.* From Latin *levis*, trifling.

Common name: Guinea-pig salivary-gland virus.

Host: *CAVIIDAE—Cavia porcellus* (L.), guinea pig (only known host; fetus more susceptible than post-natal animal, even if from immune mother).

Insusceptible species: Rabbit, rat, cat, chicken, pigeon, dog, mouse, monkey (*Macacus rhesus*).

Geographical distribution: United States, England.

Induced disease: In guinea pig, submaxillary glands show swollen epithelial cells containing relatively dense acidophilic inclusions of granular material within enlarged nuclei, especially in ducts of the serous portion of the gland, and larger but fewer intracytoplasmic inclusions; experimentally, by intracerebral injection of young guinea pig, prodromal period of about 2 days, then elevation of temperature to 105 or 106° F; a day later, hair raised, animal quiet; subsequently, irritability with tremors and slight convulsive movements; by fifth day, usually prostration, jerking movements, and ensuing death; brain shows no gross lesions but exudate over surface; in meningeal exudate, many cells each containing an acidophilic mass within its nucleus; by subcutaneous injection, virus recoverable after 2 weeks from submaxillary glands, cervical lymph nodes, kidney, and lung, not from blood, liver, or spleen.

Transmission: Experimentally, by inoculation of submaxillary gland or by intracerebral or subcutaneous injection of materials from infected glands; with difficulty from brain to brain. Pilocarpine stimulation increases numbers of inclusions.

Serological relationships: Specific neutralizing antibody is found in blood serum of animals that are carrying virus in their submaxillary glands.

Immunological relationships: Active immunity may be dependent on existence of more or less active lesions.

Thermal inactivation: At 54° C in 1 hour.

Filterability: Passes Berkefeld N filter candle.

Other properties: Viable in 50 per cent glycerine at least 11 days.

Strains: An unusually virulent strain, killing infected animals whatever the route of injection, has been described but not given a distinctive name (Rosenbusch and Lucas, Am. Jour. Path., *15*, 1939, 303–340).

Literature: Andrewes, Brit. Jour. Exp. Path., *11*, 1930, 23–34; Cole and Kuttner, Jour. Exp. Med., *44*, 1926, 855–873; Hudson and Markham, *ibid.*, *55*, 1932, 405–415; Jackson, Jour. Inf. Dis., *26*, 1920, 347–350; Kuttner, Jour. Exp. Med., *46*, 1927, 935–956; Kuttner and T'ung, *ibid.*, *62*, 1935, 805–822; Lucas, Am. Jour. Path., *12*, 1936, 933–948; Markham, *ibid.*, *14*, 1938, 311–322; Markham and Hudson, *ibid.*, *12*, 1936, 175–182; Pearson, *ibid.*, *6*, 1930, 261–274; Scott, Jour. Exp. Med., *49*, 1929, 229–236; Scott and Pruett, Am. Jour. Path., *6*, 1930, 53–70.

3. **Rabula innocuus** *spec. nov.* From Latin *innocuus*, harmless.

Common name: Hamster salivary-gland virus.

Host: *CRICETIDAE—Cricetulus griseus* M. Edw., Chinese hamster.

Insusceptible species: *MURIDAE*—rat; *Mus musculus* L., white mouse.

Geographical distribution: China.

Induced disease: In hamster, no obvious disease externally but inclusion bodies in submaxillary glands.

Thermal inactivation: At 56° C in 30 minutes.

Literature: Kuttner and Wang, Jour. Exp. Med., *60*, 1934, 773–791.

4. Rabula exiguus *spec. nov.* From Latin *exiguus*, petty.

Common name: Rat salivary-gland virus.

Host: *MURIDAE*—rat.

Insusceptible species: *MURIDAE—Mus musculus* L., mouse. *CRICETIDAE—Cricetulus griseus* M. Edw., Chinese hamster.

Geographical distribution: China, Canada.

Induced disease: In rat, no obvious disease externally, but intranuclear inclusions in cells of the submaxillary glands.

Literature: Kuttner and Wang, Jour. Exp. Med., *60*, 1934, 773–791; Thompson, Jour. Inf. Dis., *50*, 1932, 162–170.

5. Rabula latens *spec. nov.* From Latin *latens*, hidden or lurking.

Common name: Mouse salivary-gland virus.

Host: *MURIDAE—Mus musculus* L., mouse.

Insusceptible species: *MURIDAE*—rat. *CRICETIDAE—Cricetulus griseus* M. Edw., Chinese hamster. *LEPORIDAE*—rabbit. *CAVIIDAE—Cavia porcellus* (L.), guinea pig.

Geographical distribution: China, Canada, United States.

Induced disease: In mouse, no obvious disease externally, but inclusion bodies in acinar tissue of serous and mucous portions of submaxillary glands; occasionally also in duct cells or alveolar cells of parotid gland; affected cells hypertrophied. In Swiss white mice, extensive lesions in liver and spleen but emulsions of these organs fail to infect; rare pancreatic lesions.

Transmission: Experimentally, by intraglandular, subcutaneous, intraperitoneal, intratesticular or intracerebral inoculation; inclusion bodies appear in salivary glands irrespective of site of inoculation.

Thermal inactivation: At 60° C in 30 minutes.

Filterability: Passes Berkefeld V filter candle.

Literature: Kuttner and Wang, Jour. Exp. Med., *60*, 1934, 773–791; McCordock and Smith, *ibid.*, *63*, 1936, 303–310; Thompson, Jour. Inf. Dis., *58*, 1936, 59–63.

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