

FAMILY *INOVIRIDAE*

TAXONOMIC STRUCTURE OF THE FAMILY

FAMILY	<i>Inoviridae</i>
GENUS	<i>Inovirus</i>
GENUS	<i>Plectrovirus</i>

VIRION PROPERTIES

Morphology

Virions in this family are rods or filaments which contain a single-stranded circular DNA genome within a cylindrical protein shell. There are no lipid components. Virion length is determined by both the exact number of nucleotides in the DNA, usually between 5 and 10 kb, and the DNA conformation maintained by the specific

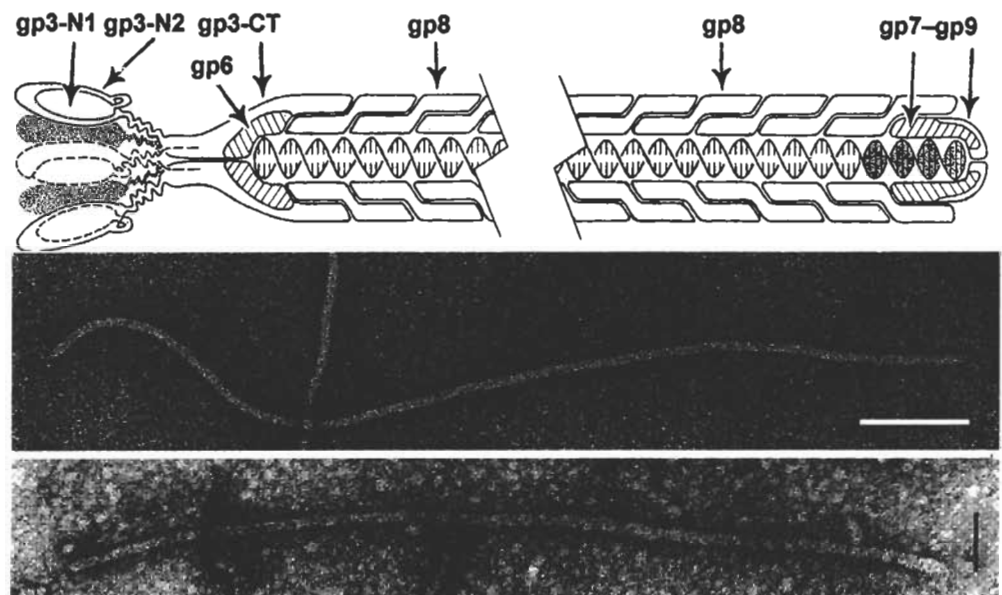


FIGURE 1

Virions of the family *Inoviridae*: (Upper) Diagram of both ends of *Inovirus* F pilus-specific coliphage (fd). The diameter is 7 nm. The circular DNA forms a right-handed two-stranded helix and has regular base pairing in the PAC site hairpin (shaded), but not elsewhere. [Adapted from Day, L. A., *et al.* (1988), Lubkowski, J. *et al.* (1998), and Rakonjac, J., *et al.* (1999).] (Center) Negative contrast electron micrograph of inovirus fd showing a tapered end with extensions due to adsorption proteins. The bar represents 100 nm. [From Gray, C. W., *et al.* (1981), courtesy of Dr. Gray.] (Lower) Negative contrast electron micrograph of a preparation of plectrovirus *Acholeplasma* phage L51 showing a rod-shaped virion and an abnormally long particle. The bar represents 50 nm. [From Maniloff, J., *et al.* (1977).]

protein shell. Virion length can change during evolution either through insertions or deletions in the genomes, as well as through mutations that alter the coat proteins. In the genus *Inovirus*, virions are all only about 7 nm in diameter, but lengths vary almost threefold, from 700 nm [*Pseudomonas phage Pf3* (Pf3)] to 2,000 nm [*Pseudomonas phage Pf1* (Pf1)]. Electron micrographs reveal different structures at the ends, one small and blunt, and the other larger and more variable. Engineered virions of Ff (the collective designation for *Enterobacteria phage M13* (M13), *Enterobacteria phage f1* (f1), and *Enterobacteria phage fd* (fd)), in which the shell protein (gp8) or the adsorption protein (gp3) are fusions with foreign peptides, are longer than wild type, due to the extra DNA, and can have other changes in shape, depending on the size and properties of the fusions. Wild-type DNA conformations can have very different spectroscopic properties and dramatically different extensions, with axial projections of the distance between neighboring nucleotides (the rise per nucleotide) ranging from a low of 0.23 nm for Pf3, over 0.27 nm for Ff, to a high of 0.61 nm for Pf1. X-ray fiber diffraction shows some capsids to have 5-fold rotational and 2-fold screw symmetry (C_5S_2), and others to have onefold rotational and 5.4- to 5.5-fold screw symmetry ($C_1S_{5.4}$). *Plectrovirus* virions are nearly straight rods with one end rounded and the other more variable. Virions of *Acholeplasma* phages are 70–90 nm long and 14–16 nm in diameter, and *Spiroplasma* phages are 230–280 nm long and 10–15 nm in diameter. Very long rods are frequently observed. Negative stained images suggest a 4 ± 2 nm hollow core. Optical diffraction of images (*Acholeplasma phage MV-L1* (L1)) suggests morphological units arranged in 2-fold rotational and 5.6-fold screw symmetry ($C_2S_{5.6}$). DNA conformations in this genus have apparent rise-per-nucleotide values in the range 0.02 to 0.03 nm (Fig. 1).

Physicochemical and Physical Properties

Within the family, virions are sensitive to chloroform and are generally resistant to heat and a wide range of pH. For the genus *Inovirus*, the buoyant densities in CsCl are 1.29 ± 0.01 , and DNA contents range from 6% to 14%. The M_r range from 12 to 34×10^6 , almost a threefold range, whereas the S_{20w} fall in a narrow range, 41–44S; the closely similar sedimentation rates are largely determined by closely similar mass per length. Translational and rotational diffusion constants are consistent with essentially rigid rods, yet *Inovirus* virions can bend considerably without breaking. Two species (*Enterobacteria phage C2* (C2) and *Enterobacteria phage X* (X)) appear to be more flexible than the others according to electron microscopy. Spectroscopic measurements reveal similar protein conformations, all highly helical, but various DNA conformations, some base-stacked and some not. For the genus *Plectrovirus*, buoyant densities of 1.39 g/cm^3 in CsCl and 1.21 g/cm^3 in metrizamide were reported for *Spiroplasma phage 1* (SpV1).

Nucleic Acid

Virions contain one molecule of infectious, circular, positive sense ssDNA. *Inovirus* genomes range from 6 to 9 kb. *Plectrovirus* genomes are 4.5 kb for *Acholeplasma* phages and about 8 kb for *Spiroplasma* phages. DNA sequences of *Inovirus* species fd, M13, f1, If1 (*Enterobacteria phage If1*), IKe (*Enterobacteria phage IKe*), I2-2 (*Enterobacteria phage I2-2*), Pf1, Pf3, Cf1c (*Xanthomonas phage Cf1c*) (and Cf1t; *Xanthomonas phage Cf1t*), and *Vibrio phage fs1* (fs1), as well as *Plectrovirus* species *Spiroplasma phage 1-R8A2B* (SpV1-R8A2B) and L1 are available in the GenBank or EMBL databases.

Proteins

In genus *Inovirus*, Ff (M13, f1, fd) virions, the long shells are composed of 2700 copies of gp8 ($Mr\ 5.2 \times 10^3$), the adsorption end has several copies (probably 5 each) of gp3 ($Mr\ 43 \times 10^3$) and gp6 ($Mr\ 12 \times 10^3$), and several copies (probably five each) of gp7 ($Mr\ 3.5 \times 10^3$) and gp9 ($Mr\ 3.3 \times 10^3$) form the assembly nucleation end. Six nonstructural proteins have been identified: morphogenetic proteins gp1 ($Mr\ 35 \times 10^3$), gp11 ($Mr\ 8 \times 10^3$), and gp4 ($Mr\ 50 \times 10^3$), and DNA replication proteins gp2 ($Mr\ 46 \times 10^3$), gp10 ($Mr\ 12 \times 10^3$), and gp5 ($Mr\ 9.8 \times 10^3$). In genus *Plectrovirus* L1 and L51 virions, the major capsid protein is probably of $Mr\ 19 \times 10^3$, a protein with a strong tendency to aggregate, and there is at least one minor protein. The genome has only four ORFs. In genus *Plectrovirus* SpV1-R8A2B and *Spiroplasma phage 1-T78* (SpV1-T78) the major capsid protein is of $Mr\ 7.5 \times 10^3$.

Lipids

None reported.

Carbohydrates

None reported.

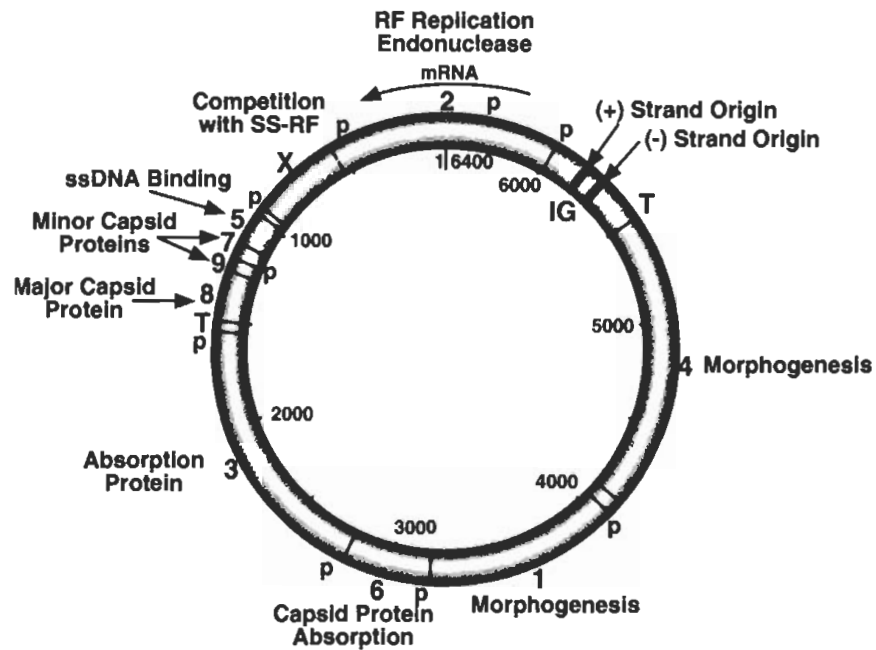
GENOME ORGANIZATION AND REPLICATION

Genomes replicate either independently, by the rolling-circle mechanism like free plasmids, or with the chromosome if the viral genome becomes integrated. In the normal productive infectious cycle there are five steps: phage adsorption and uptake of the infecting ssDNA circle, conversion of the ssDNA circle to a parental replicative form (RF) by host cell enzymes, semiconservative RF replication initiated by a viral endonuclease, synthesis of progeny ssDNA sequestered by ssDNA binding protein, and the membrane based-assembly process that extrudes progeny virions into the medium. Genome organization reflects this sequence in that viral genes for DNA replication, for virion structure, and for virion morphogenesis are grouped in succession around the circle (Fig. 2).

The most detailed studies have been done on *Inovirus* Ff (M13, f1, and fd). In each of the other systems examined, closely similar or parallel phenomena have been observed. Genes are closely spaced; several genes are translated from overlapping reading frames or from alternate starts in the same frame. Intergenic regions contain the complementary and viral strand replication origins and DNA packaging signals. Phage adsorption involves specific interaction between the F pilus and a domain of gp3 on the infecting phage, and ssDNA translocation into the cytoplasm involves specific interactions between another domain of gp3 with other host membrane proteins. The ssDNA is converted to a supercoiled dsDNA replicative form (RF) by cellular enzymes. Phage DNA replication begins when the viral endonuclease gp2 expressed from parental RF nicks this RF at a specific, high-symmetry site. Progeny RF produced via ssDNA intermediates in rolling-circle replication become templates for further RF replication and further mRNA synthesis. Gp10 and gp5 can downregulate the nicking activity of gp2. When sufficient gp5 is made, complementary strand synthesis is blocked and complexes of gp5 and progeny viral ssDNA accumulate. Assembly is initiated at the membrane by concerted interactions of gp7,

FIGURE 2

Genetic map of *Inovirus F* pilus-specific coliphages showing functions of gene products. Not shown are the locations of gene 10 (DNA replication) and gene 11 (morphogenesis); these are the C-terminal one-thirds of gp2 and gp1, respectively, both derived translationally. DNA replication origins in intergenic region (IG) are shown. P = promoter, T = transcription terminator. [From Kornberg, A., Baker, T. A. (1991). *DNA Replication*, 2nd ed. New York: WH Freeman and Co., p. 561; see Hill *et al.* (1991) for comparisons of the Ff, Pf3, and Pf1 genomes, and an alternate map numbering convention.]



gp9, gp1, and gp11 and a specific packaging signal in a hairpin on the ssDNA in the gp5 complex. Assembly proceeds at the inner membrane where about 1500 subunits of gp5 are displaced by 2700 subunits of gp8. Both gp1 and gp11 appear to be involved in this transfer of the DNA from gp5-ssDNA complexes into the assembling virion. Gp1 may also function in the formation of adhesion zones between the inner and outer membranes by interacting with outer membrane pores formed by subunits of viral protein gp4. Assembly of virions is completed by addition of gp6 and gp3. There are notable exceptions to this overall pathway. Lysogenic strains encode integrases, and viral sequences, partial or complete, are found integrated at several chromosomal sites. In Cf1t and Cf16 (*Xanthomonas phage Cf16*) variants of Cf1c, the first *Inovirus* lysogens characterized, the gene for site specific integration shows no homology with any Ff genes. Another species with a lysogenic phase is the *Vibrio phage CTX* (CTX), the genome of which encodes the two subunits of cholera toxin. Upon conversion of the lysogen to the nonlytic productive cycle the toxin genes become highly expressed and the toxin is released. The *Plectrovirus* species *Acholeplasma phage MV-L51* (L51) has been shown to be similar to this scheme with respect to DNA replication pathways, and virions are assembled at the membranes as they are released into the medium without lysis. This is presumably true for all species in the genus replicating as independent plasmids and producing virus by extrusion. The genomes of two viruses (L1 and SpV1) have both been found integrated into the host chromosome at one or more sites.

BIOLOGICAL PROPERTIES

Members of the family infect their natural hosts without causing lysis, and the infected cells continue to divide and produce virus indefinitely. The hosts are plant and animal pathogens. In several systems the phage enter into lysogenic phases.

Cell growth rates are slowed marginally by infection. On plates the slower growth usually allows the formation of turbid plaques. Sometimes there is phage multiplication but no plaque formation. *Inovirus* hosts are all gram-negative bacteria (i.e., *Escherichia coli*, *Salmonella*, *Pseudomonas*, *Vibrio*, *Xanthomonas*, etc.). Host ranges are determined primarily by host cell receptors, which are usually conjugative pili. Some pili are encoded chromosomally and some are encoded on plasmids of different incompatibility groups, i.e., phage Ff (M13, f1, fd) adsorbs to IncF pili, Pf3 to IncP pili, tf-1 to IncT pili, X to IncX pili, etc. Transmission of the plasmids to new bacterial species usually transfers phage sensitivity. Additional host range determinants include restriction–modification systems, host periplasmic proteins involved in viral ssDNA translocation into the cytoplasm, and host protein(s) involved in membrane assembly. Transfections of non-natural hosts with naked ssDNA or dsDNA are sometimes possible. When *Vibrio cholera* phage lysogens colonize the human intestine, states of elevated cholera toxin expression and release, and of progeny filamentous cholera phage extrusion, are induced. Thus *Inovirus* lysogeny is a critical virulence factor in cholera pathogenesis. *Plectrovirus* species infect wall-less *Acholeplasma* and *Spiroplasma* and their receptors may contain both polysaccharide and protein components, but they are not well characterized. The indications of lysogeny in the L1 and SpV1-R8A2B systems suggest that the two potential modes of carrier states, as free plasmids with virus extrusion or as lysogens, might be generally true for all members of the Family *Inoviridae*.

GENUS *INOVIRUS*

Type Species *Enterobacteria phage M13* (M13)

DISTINGUISHING FEATURES

Infectivity is sensitive to sonication; ether sensitivity is variable. Nucleic acid is 6–14% by weight of particle, and G + C is 40–60%. Sedimentation rates are $42 \pm 2S$. Virions have no carbohydrate. Host range is certain genera in the gamma-purple phylogenetic branch of gram-negative bacteria, i.e., *Enterobacteria*, *Pseudomonas*, *Vibrio*, and *Xanthomonas*.

LIST OF SPECIES DEMARCATION CRITERIA IN THE GENUS

Demarcation criteria in the genus are:

- Particle length,
- Host range
- Capsid symmetry
- Antigenic properties, and
- DNA conformation and ratios of nucleotides per subunit

An individual species in this genus is distinguishable by its host range, major coat protein sequence, and capsid symmetry. For example, the species Ff includes strains fd, f1, and M13 which have the same host range, capsid symmetry, and coat sequence (except for a single amino acid; their DNA sequences differ by less than ~1.5%). Although mutations, natural or otherwise, might dramatically affect virion length, ratios of nucleotides to subunits, DNA conformation, and antigenic properties of these strains (as in phage display work), species boundaries are not likely to be crossed. The viruses If1, IKe, and I2-2 have the same capsid symmetry and very similar sequences, but their host ranges differ, so they are different species.

LIST OF SPECIES IN THE GENUS

Official virus species names are in italics. Tentative virus species names, alternative names (), strains, or serotypes are not italicized. Virus names, , their host ranges { }, genome sizes (in kb), genome sequence accession numbers [], and assigned abbreviations () are:

Species in the Genus

1-Phages of *Enterobacteriaceae*:

<i>Enterobacteria phage If1</i>	{ <i>E. coli</i> , <i>S. typhimurium</i> ; Incl}	8.5 kb	[NC001954]	(If1)*
<i>Enterobacteria phage IKe</i>	{ <i>E. coli</i> ; Inc I ₂ , N, P-1}	6.9 kb	[X02139]	(IKe)*
<i>Enterobacteria phage I₂-2</i>	{ <i>E. coli</i> ; Inc I ₂ }	6.7 kb	[X14336]	(I ₂ -2)*
<i>Enterobacteria phage M13</i>	{ <i>E. coli</i> IncF, Hfr}	6.4 kb	[V00604]	(M13, Ff)*
Enterobacteria phage fl	{ <i>E. coli</i> IncF, Hfr}	6.4 kb	[J02448]	(f1, Ff)*
Enterobacteria phage fd	{ <i>E. coli</i> IncF, Hfr}	6.4 kb	[V00602]	(fd, Ff)*
Enterobacteria phage AE2	{ <i>E. coli</i> IncF (Hfr)}			(AE2)
Enterobacteria phage δA				(probably all Ff)
Enterobacteria phage Ec9				
Enterobacteria phage HR				
Enterobacteria phage ZJ/2				
<i>Enterobacteria phage X-2</i>	{ <i>E. coli</i> , <i>S. typhi</i> , <i>Sr. marc.</i> ; IncX, unique R775}			(X-2)
<i>Enterobacteria phage C-2</i>	{ <i>E. coli</i> , <i>S. typhi</i> ; IncC}	8.1 kb		(C-2)
<i>Enterobacteria phage X</i>	{ <i>E. coli</i> , <i>S. typhi</i> , <i>Sr. marc.</i> , others; IncX, I ₂ , N, P-1, others}			(X)
<i>Enterobacteria phage PR64FS</i>	{ <i>E. coli</i> ; IncR}			(PR64FS)
<i>Enterobacteria phage SF</i>	{ <i>E. coli</i> , <i>K. pneumoniae</i> , <i>S. typhi</i> , others; IncS}			(SF)
<i>Enterobacteria phage tf-1</i>	{ <i>E. coli</i> , <i>S. typhi</i> ; IncT}			(tf-1)

2- Phages of Spirillaceae:

<i>Vibrio phage</i> 493	{ <i>V. cholera</i> 0139-Aj27, El Tor}	9.3 kb		(493)
<i>Vibrio phage</i> fs1	{ <i>V. cholera</i> 01, 0139}	6.3 kb	[D89074]	(fs1)
<i>Vibrio phage</i> fs2	{ <i>V. cholera</i> 01}	8.5 kb		(fs2)
<i>Vibrio phage</i> CTX	{ <i>V. cholera</i> 0395, Peru 15, El Tor, others; lysogenic}	7 kb	[AF09912, AF053180, U83795,-6]	(CTX)
<i>Vibrio phage</i> v6	{ <i>V. parahaemolyticus</i> }			(v6)
<i>Vibrio phage</i> Vf12	{ <i>V. parahaemolyticus</i> }	8.0kb		(Vf12)
<i>Vibrio phage</i> Vf33	{ <i>V. parahaemolyticus</i> }	8.0kb		(Vf33)
<i>Vibrio phage</i> VSK	{ <i>V. cholera</i> 0139-B04, lysogenic <i>V. cholera</i> 0139-P07, normal}			(VSK)

3-Phages of Pseudomonadaceae:

<i>Pseudomonas phage</i> Pf1	{ <i>P. aeruginosa</i> PAK}	7.3 kb	[X52107]	(Pf1)*
<i>Pseudomonas phage</i> Pf2	{ <i>P. aeruginosa</i> PAK}			(Pf2)
<i>Pseudomonas phage</i> Pf3	{ <i>P. aeruginosa</i> PAO; IncP-1}	5.8 kb	[M11912]	(Pf3)*

4-Phages of Xanthomonadaceae:

<i>Xanthomonas phage</i> Cf1c	{ <i>X. camp. citri</i> : normal}	7.3 kb	[M57538, U41819]	(Cf1c)
<i>Xanthomonas phage</i> Cf1t	{ <i>X. camp. citri</i> : lysogenic}		[U08370]	(Cf1t)
<i>Xanthomonas phage</i> Cf16	{ <i>X. camp. citri</i> : neolysogenic}			(Cf16)
<i>Xanthomonas phage</i> Cf1tv	{ <i>X. camp. citri</i> -Cf1t : lytic}			(Cf1tv)
<i>Xanthomonas phage</i> Lf	{ <i>X. campestris</i> pv. <i>campestris</i> : lysogenic}	6.0 kb	[X70327-31, U10884, U38235, AF018286]	(Lf)
<i>Xanthomonas phage</i> Xf	{ <i>X. campestris</i> pv. <i>oryzae</i> }	7.4 kb		(Xf)*
<i>Xanthomonas phage</i> Xfo	{ <i>X. oryzae</i> pv. <i>oryzae</i> }	7.6 kb		(Xfo)
<i>Xanthomonas phage</i> Xfv	{ <i>X. campestris</i> pv. <i>vesicatoria</i> }	6.8 kb		(Xfv)

An asterisk after the abbreviation in bold indicates that capsid symmetry has been assigned; for those in group 1 it is C_5S_2 ; for those in groups 2 and 4 it is $C_1S_{5.4}$. *E. coli*: *Escherichia coli*, *S. typhi*: *Salmonella typhi*, *Sr. marc.*: *Serratia marcescens*, *K. pneumoniae*: *Klebsiella pneumoniae*, *V. cholera*: *Vibrio cholera*, *V. parahaemolyticus*: *Vibrio parahaemolyticus*, *P. aeruginosa*: *Pseudomonas aeruginosa*, *X. camp. citri*: *Xanthomonas campestris citri*, *X. oryzae*: *Xanthomonas oryzae*.

Tentative Species in the Genus

None.

GENUS *PLECTROVIRUS*

Type Species *Acholeplasma phage MV-L51* (L51)

DISTINGUISHING FEATURES

Virions are resistant to nonionic detergents (Noridiet P-40 and Triton X-100) and slightly sensitive to ether. Genome of *Spiroplasma phage 1* (SpV-1) is 23% G + C. No data on carbohydrates have been reported. Adsorption is to cell membrane of wall-less mycoplasma host cells. Host range of the *Acholeplasma phage MV-L51* (L51) is some *Acholeplasma laidlawii* strains and of SpV-1 is some *Spiroplasma citri* strains.

LIST OF SPECIES DEMARCATION CRITERIA IN THE GENUS

Plectrovirus species differ in host range.

LIST OF SPECIES IN THE GENUS

Official virus species names are in italics. Tentative virus species names, alternative names (), strains, or serotypes are not italicized. Virus names, their host ranges { }, genome sizes (in kb), genome sequence accession numbers [], and assigned abbreviations () are:

Species in the Genus

1-Phages of <i>Acholeplasma</i> :				
<i>Acholeplasma phage MV-L51</i>	{ <i>A. laidlawii</i> }	4.5 kb		(L51)
2-Phages of <i>Spiroplasma</i>				
<i>Spiroplasma phage 1-KC3</i>	{ <i>S. melliferum</i> BC3}			(SpV1/KC3)
<i>Spiroplasma phage 1-aa</i>	{ <i>S. citri</i> -SP-V3}	8.5 kb		(SpV1-aa)
<i>Spiroplasma phage 1-R8A2B</i>	{ <i>S. citri</i> Morocco}	8.3 kb	[X51344]	(SpV1-R8A2B)
<i>Spiroplasma phage 1-C74</i>	{ <i>S. citri</i> Corsica}	7.8 kb		(SpV1-C74)
<i>Spiroplasma phage 1-T78</i>	{ <i>S. citri</i> Turkey}	8.5 kb		(SpV1-T78)
<i>Spiroplasma phage 1-S102</i>	{ <i>S. citri</i> Syria}	6.9 kb		(SpV1-S102)

A. laidlawii: *Acholeplasma laidlawii*, *S. melliferum*: *Spiroplasma melliferum*, *S. citri*: *Spiroplasma citri*.

Tentative Species in the Genus

<i>Acholeplasma phage MV-L1</i>	{ <i>A. laidlawii</i> : lysogenic}	4.5 kb	[X58839]	(L1)
<i>Acholeplasma phage MV-G51</i>	{ <i>A. laidlawii</i> }			(G51)
<i>Acholeplasma phage 0c1r</i>				(0c1r)
<i>Acholeplasma phage 10tur</i>				(10tur)
<i>Spiroplasma phage C1/TS2</i>				(C1/TS2)

LIST OF UNASSIGNED VIRUSES IN THE FAMILY

None reported.

PHYLOGENETIC RELATIONSHIPS WITHIN THE FAMILY

Not available.

SIMILARITY WITH OTHER TAXA

None reported.

DERIVATION OF NAMES

Ino: from Greek *nos*, "muscle."

Plectro: from Greek *plektron*, "small stick."

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CONTRIBUTED BY

Day, L. A., and Maniloff, J.