Accession number: txt002sdr; **Accession date:** 18 November 1997; **Last update:** 18 November 1997
Archived previous version: no; **Replaced by revised version**: no

Human herpesvirus 7

Abbreviations: HHV-7

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Human herpesvirus 7 (HHV-7) is a recently described T-lymphotropic herpesvirus, which infects almost all children by the age of three years and persists lifelong, with the shedding of infectious virus in saliva. HHV-7 is similar to human herpesvirus 6 (HHV-6) in its genetic content and in many of its biological properties, which include the ability to cause at least some cases of exanthem subitum (roseola). Despite these similarities, important differences between HHV-7 and HHV-6 exist, including the fact that HHV-7 binds to the cellular CD4 molecule and uses this protein as a necessary component of its receptor, while HHV-6 binds to a different (and unknown) receptor. Furthermore, the pathogenesis and sequelae of HHV-7 infection remain very poorly understood. This review provides a critical summary of research on HHV-7.

HHV-7 was isolated in 1990 from the peripheral blood of a healthy individual whose $CD4^+$ T lymphocytes (T cells) underwent a spontaneous cytopathic effect in tissue culture (Ref. 1). Subsequently, the virus was isolated from phytohaemagglutinin (PHA)-activated peripheral blood mononuclear cells (PBMCs) and from the saliva of healthy adults; the virus could not, however, be isolated from resting PBMCs. Taken together, these findings are consistent with the following conclusions: (1) that HHV-7 might be present in a latent state in normal PBMCs and (2) that it is constitutively shed in saliva, and possibly transmitted by this route (Refs 2, 3, 4, 5, 6).

Classification

HHV-7 is most closely related to HHV-6 at the genetic level, among all known herpesviruses, and its next closest sibling is human cytomegalovirus (HCMV). HHV-7 has not, as yet, been classified,

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although its genetic and biological properties would seem to be consistent with a similar classification proposed for HHV-6, which is in the genus *Roseolovirus*, within the beta herpesvirus subfamily.

Lifecycle

The cell-infection and genetic properties of HHV-7 are discussed below, and are summarised (along with those of HHV-6) in Table 1 (tab001sdr).

Host-cell tropism

HHV-7 is tropic for primary, human T cells, but appears to have a considerably narrower *in vitro* host-cell range than HHV-6. Thus, HHV-7 infection is limited strictly to cultured primary $CD4^+$ T cells and the SupT1 cell line (reviewed in Ref. 7), whereas HHV-6 infects a wide array of cultured cells and cell lines (see the review article on HHV-6, txt001sdr). It is particularly striking that HHV-7 fails to productively infect many $CD4^+$ cells or cell lines, including primary, monocyte-derived macrophages (Ref. 8) and commonly used $CD4^+$ T cell lines, such as Jurkat and H9 (Ref. 9).

Receptor interactions

The early events involved in the infection of cells by the prototypic human herpesvirus, herpes simplex virus type 1 (HSV-1) are believed to include an initial interaction with cell-surface heparan sulfate proteoglycans (HSPGs), which is then followed by binding to a specific cellular receptor (Ref. 10). Events involved in HHV-7 infection are not well characterised, but include the specific binding of the virus to the cellular CD4 molecule (Ref. 11).

As is the case for human immunodeficiency virus type 1 (HIV-1), CD4 expression is necessary but not sufficient for infection of T cells by HHV-7. Thus, HHV-7 is unable to penetrate many $CD4^+$ cells, including HeLa-CD4 cells (Ref. 11). This suggests that factor(s), in addition to CD4, are required for infection of the cell with the virus; these might include the presence of cell-surface proteoglycans. Highly sulfated HSPGs that are capable of binding to the glycoprotein B (gB) of HHV-7 are present on SupT1 cells but not on other $CD4^+$ T-cell lines (such as Jurkat and PM1) or HeLa cells, none of which is susceptible to HHV-7 infection (Ref. 12). Furthermore, *de novo* expression of CD4 in several cell lines of haematopoietic lineage, including K562 cells, has been shown to render these cells fully permissive for HHV-7 infection (Ref. 13); this suggests that some (for example K562) but not all (for example HeLa) cells express the putative co-receptor(s) required for HHV-7 entry.

Effect on T cells

HHV-7 productively infects $CD4^+$ T cells, inducing a cytopathic effect (CPE) that is similar to the CPE induced by HIV-1 and by HHV-6. This CPE is characterised by membrane blebbing and the presence of multinucleated giant cells (syncytia) (Refs 1, 14). However, HHV-7 differs from HHV-6 in its effects on the expression of immunoregulatory T-cell surface proteins.

HHV-7 has no effect on the cellular expression of the CD3 cell-surface molecule, but causes a dramatic down-regulation of CD4 expression, within 6–9 days after infection (Refs 15, 16). This down-modulation of CD4 antigen appears to occur via several complementary pathways, much like the inhibition of cell-surface CD4 expression that occurs in HIV-1-infected cells. Thus, HHV-7 infection has been shown to result in a decline in the total amount of CD4 protein and mRNA in SupT1 cells, and infection also induces post-translational effects on CD4 expression in primary T cells and, to a lesser extent, in SupT1 cells (Refs 15, 16).

Genetics and molecular biology

The linear, double-stranded DNA genome of HHV-7 has been completely sequenced, and is 145 kilobases (kb) in length (Ref. 17). The viral genome is co-linear with the HHV-6 genome throughout its entirety; in common with HHV-6, it shares several important genetic properties with the alpha herpesvirus family (see below).

The HHV-7 genome is approximately 10% shorter than the HHV-6 genome, and consists of a long unique region (approximately 133 kb), which is bounded on both sides by direct repeat elements (DRs; approximately 6 kb each). The overall arrangement of the HHV-7 genome is, therefore, identical to that of HHV-6, which is shown schematically in Figure 1 (fig005sdr). Additional similarities in genome structure include the presence of human telomeric repeat sequence (TRS) motifs near, but not at, the genome ends of both viruses, although the function of these elements remains unknown (Ref. 18).

Localised regions of divergence between the genomes of HHV-7 and HHV-6 also occur. These

https://doi.org/10.1017/S1462399497000112 Published online by Cambridge University Press

Accession number: txt002sdr; **Accession date:** 18 November 1997; **Last update:** 18 November 1997
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Table 1. Genetics and cell infection of human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7) (tab001sdr)

	HHV-6	HHV-7
Tropism	T-lymphotropic. A broad host-cell range can be infected *in vitro* (particularly for HHV-6A), which includes primary T cells, NK cells and astrocytes, as well as continuous cell lines of T cells, B cells, megakaryocyte and glial lineages.	T-lymphotropic. Tropism is restricted to: $CD4^+$ T cells and the SupT1 cell line
Variants	HHV-6A, HHV-6B. HHV-6A might have a different host-cell range/pathogenesis and is ~5% divergent at the nucleotide level from HHV-6B.	None known.
Receptor (for binding to cells and entry)	Unknown. The broad host-cell tropism of HHV-6 suggests that either (1) the receptor is ubiquitous or (2) more than one molecule can be used for virus entry.	CD4 is a necessary receptor component, but is not 'sufficient' for virus entry.
Cell markers	HHV-6 can induce expression of CD4 on some $CD4^-$ cells.	HHV-7 induces loss of cell-surface CD4.
Monocyte infection	HHV-6 can persist in monocytes.	HHV-7 does not infect monocytes.
Genome size	U1102 strain of HHV-6A is 159 kb with DRs of 8 kb.	JI strain of HHV-7 is 145 kb with DRs of 6 kb.
AAV-2 rep gene homologue	ORF U94 is a homologue of AAV-2 *rep*.	No homologue of AAV-2 *rep*.
Unique virion glycoprotein	Encodes gp105, a viral glycoprotein that derives from a highly spliced mRNA spanning multiple ORFs at the right end of the unique segment of the HHV-6 genome (U96-100). gp105 is a major virion component and contains a major neutralising antibody epitope.	Predicted to encode a homologue of gp105.
	Shared properties of HHV-6 and HHV-7	
Replication	In PBMCs, replication is slow and lytic; syncytia are induced.	
Genetics and viral genome	HHV-6 and HHV-7 are closely related and co-linear. Telomeric DNA is present near (not at) the genomic termini. They share a common *ori*Lyt organisation, similar to HSV-1 *ori*S. They encode: (1) U73: a homologue of the HSV-1 origin-binding protein; (2) U12 and U51: two homologues of cellular G-protein-coupled receptors; (3) U20 and U85: two genes with homology to genes of the immunoglobulin family (U85 is homologous to OX-2, a molecule that mediates a recently described T-cell co-stimulatory pathway); (4) gB, gH and gL: homologues of three of the four essential virion glycoproteins in HSV-1. They do not encode: (1) a homologue of gD, the receptor-binding HSV-1 glycoprotein.	

Abbreviations used: AAV = adeno-associated virus; DR = direct repeat; HHV = human herpesvirus; HSV-1 = herpes simplex virus type 1; kb = kilobase; NK = natural killer; ORF = open reading frame; PBMCs = peripheral blood mononuclear cells; Other details, including citations, are described in the main text.

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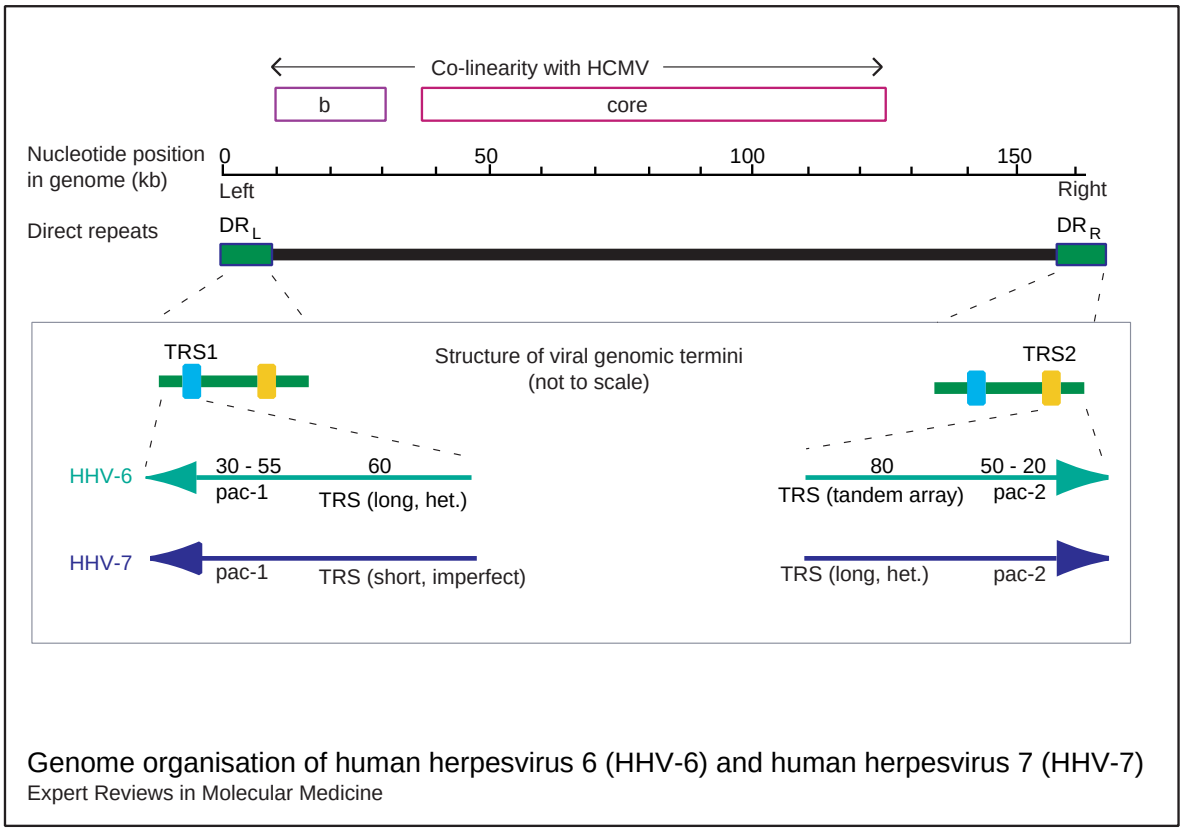

Figure 1. Genome organisation of human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7). The overall genome organisation of HHV-6 is shown, and the structure of the genomic termini of HHV-6 and HHV-7 is also presented. Numbers in the upper portion of the figure refer to nucleotide position, in kilobases (kb), within the HHV-6 genome, while numbers in the lower portion of the figure refer to nucleotide position, in base pairs (bp), relative to the viral genomic terminus (left or right), which is indicated by the arrowheads. The region of co-linearity between HHV-6 and human cytomegalovirus (HCMV) is indicated at the top of the figure, and the conserved sequence blocks (b, core) are discussed in the main text. Other indicated DNA motifs include the viral direct repeats (DR), located at the left (L) and right (R) genome ends, as well as consensus sequence motifs (pac-1, pac-2) involved in the cleavage and packaging of replicated viral DNA. TRS motifs refer to blocks of human telomeric repeat sequences ([GGGTTA]n). Distinct iterations of these motifs occur close to the genome ends of HHV-6 and HHV-7 (TRS1, TRS2), including long, heterogeneous arrays (long, het.), perfect tandem repeats (tandem array) or short, imperfect arrays (short, imperfect) **(fig005sdr).**

map to a number of loci, including (1) the viral origins of DNA replication (see below), (2) genes encoding putative viral transactivator proteins, and (3) the DR elements, which specify open reading frames (ORFs) of different coding capacities and structures in HHV-7 compared with HHV-6 (Ref. 17). In addition, the region at the right (vs. left) end of the unique component of the HHV-7 genome is significantly different from its counterpart in HHV-6. In HHV-6, this region specifies ORFs that include U94, U96 and U97, none of these is found in HHV-7 (tab002sdr, in the html version only). The result is that HHV-7 lacks the adeno-associated virus type 2 (AAV-2) *rep* gene homologue found in HHV-6 (ORF U94), as well as two of the spliced exons (ORFs U96, U97) that encode portions of the major HHV-6 virion glycoprotein, gp105 (Refs 17, 19).

Viral origins of DNA replication

The origins of lytic-phase DNA replication (*ori*Lyt) in HHV-7, HHV-6 and HCMV are all co-linear, and are located in an intergenic region immediately downstream of the gene coding for the major DNA binding protein (U41 in HHV-7). Despite this common location, the origins of HHV-7 and of HHV-6 possess a structure that is quite different from the HCMV *ori*Lyt element,

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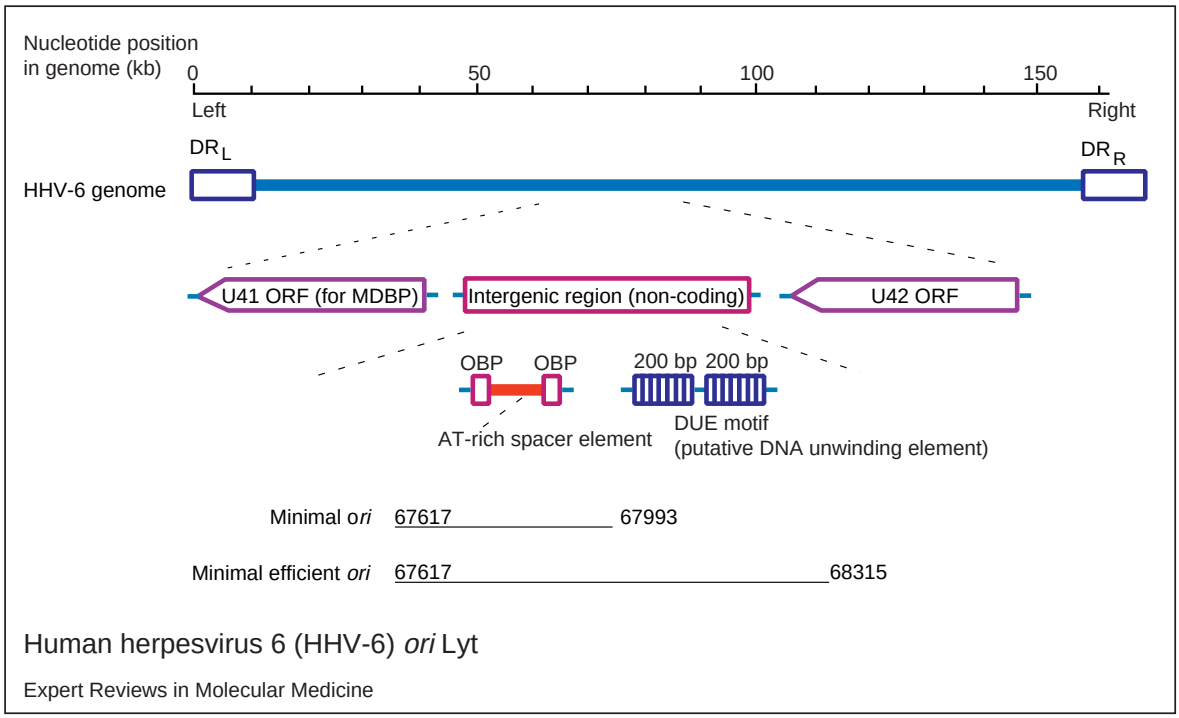

Figure 2. Schematic diagram of human herpesvirus 6 (HHV-6) *ori*Lyt. The location and structural arrangement of the HHV-6 *ori*Lyt element are shown; all numbers refer to the nucleotide position within the HHV-6 genome. As indicated in the upper portion of the figure, HHV-6 *ori*Lyt is located in an intergenic region between the U41 and U42 open reading frames (ORFs) of the virus (U41 encodes the putative major DNA-binding protein of the virus, MDBP). The intergenic region, containing the origin, is shown in detail in the lower portion of the figure. This region includes two binding sites for the HHV-6 origin-binding protein (OBP), separated by an adenine–thymine (AT)-rich spacer. Together, these elements comprise the minimal essential origin element (minimal *ori*). Immediately adjacent to this domain lie two copies of a repetitive sequence block of roughly 200 base pairs in length, which corresponds to a putative DNA unwinding element (DUE). This larger domain (minimal efficient *ori*) replicates with markedly higher efficiency than the minimal essential origin element **(fig002sdr).**

and much more similar to that of the origins of DNA replication of alpha herpesviruses. Thus, the functionally defined, minimal replication origin from HHV-7 contains two putative binding sites for a virally encoded origin-binding protein (OBP) (Ref. 20), as well as an adjacent adenine–thymine (AT)-rich element. This AT-rich element is believed to facilitate unwinding of the DNA helix during the initiation of viral DNA replication, thereby allowing replication proteins (such as the viral polymerase and helicase-primase complex) to enter the origin and begin the process of DNA synthesis.

The basic organisation of the minimal HHV-7 *ori*Lyt element is very similar to its HHV-6 counterpart (Fig. 2, fig002sdr), although there appears to be no DNA unwinding element (DUE) in the HHV-7 *ori*Lyt (Refs 20, 21). The functional significance of this difference between HHV-7 and HHV-6 remains unclear.

Unique or unusual viral genes

Genes associated with viral DNA replication

HHV-7, in common with HHV-6, encodes homologues of the genes that encode the seven essential HSV-1 replication proteins (including OBP); it also encodes homologues of HCMV genes (UL84 and UL112/UL113, Refs 17, 22, 23) that are implicated in DNA replication (see review article on HHV-6, txt001sdr). HHV-7 differs from HHV-6 in that it does not possess a homologue of the AAV-2 *rep* gene (Ref. 17), although the significance of this difference, and its implications for viral DNA replication, remain uncertain.

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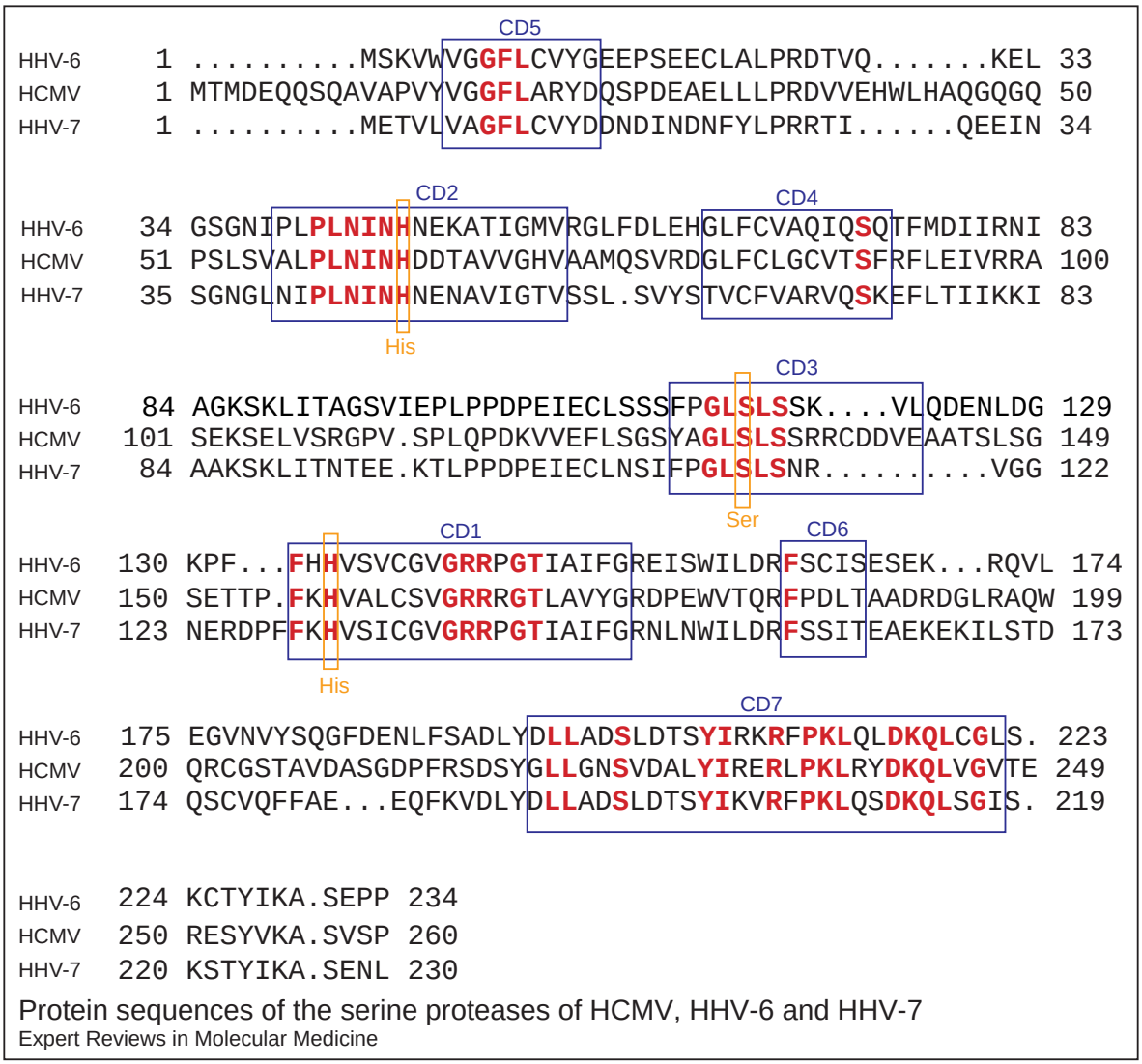

Figure 3. Alignment of the protein sequences of the serine proteases of human cytomegalovirus (HCMV), human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7). The sequence motifs in the boxes represent the conserved domains within herpesvirus proteases that have been previously described in the main text. Within these boxed regions, the residues shown in red/bold represent amino acids that are shared by HCMV, HHV-6 and HHV-7. The three active-site residues are 'boxed' in orange (in HCMV: Ser132, His63 and His157; in HHV-6: Ser116, His46 and His135; in HHV-7: Ser115, His47 and His131). Overall, at the amino acid level, the HHV-6 protease is 42% identical to HCMV protease, and the HHV-6 protease is 60% identical to the HHV-7 protease **(fig004sdr)**.

Viral glycoproteins

HHV-7 encodes homologues of all of the putative glycoprotein genes found in HHV-6, except for HHV-6 ORF U22, which has no known function (tab002sdr, in html version only). The gene product(s) responsible for virus binding to the cellular CD4 molecule remain unknown, although studies aimed at identifying this protein are ongoing in several laboratories.

Other viral genes

In common with HHV-6, HHV-7 contains two ORFs (U12, U51) that encode putative G-protein-coupled receptors, as well as ORFs that encode a phosphotransferase/ganciclovir kinase, a ribonucleotide reductase and a protease (tab002sdr, in html version only). The viral protease is likely to prove to be a useful target for antiviral drugs, because similar or related serine

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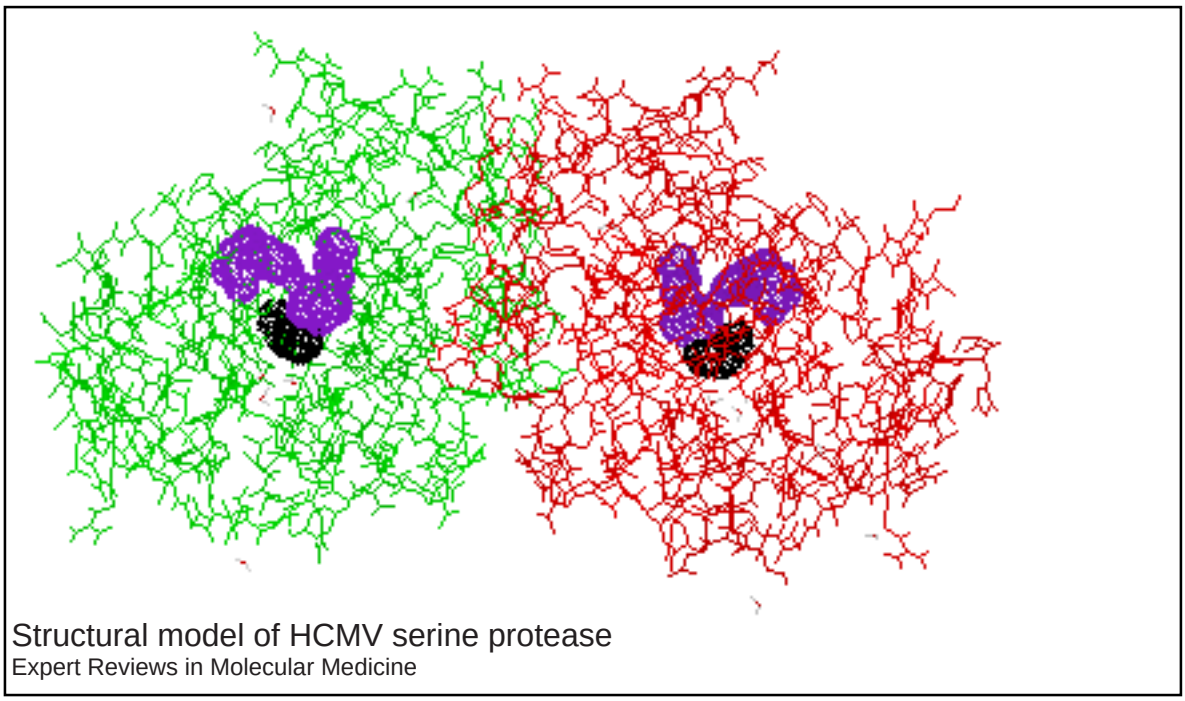

Figure 4. Structural model of human cytomegalovirus (HCMV) serine protease. The individual protease monomers within the homodimeric enzyme are coloured red and green. The catalytic triad representing the active site of the enzyme is represented by van der Waal's dot surfaces around its component atoms (to help visualise them) and is coloured black (Ser132; the catalytic nucleophile) or purple (His63, His157). This molecular representation was generated using RasMol software (http://www.glaxowellcome.co.uk/netscape/software), using the PDB database file 1CMV.PDB (http://www.pdb.bnl.gov/cgi-bin/pdbmain); residues within the protease backbone are shown in the 'wireframe' format **(fig007sdr)**.

proteases found in other herpesviruses mediate essential proteolytic processing events during viral capsid maturation (see review article on HHV-6, txt001sdr).

The predicted HHV-7 protease gene product exhibits an overall level of 60% amino acid identity to its counterpart in HHV-6, and 38% identity to the HCMV protease. Conserved sequence motifs include the His–Ser–His catalytic triad of the HCMV protease, represented by residues Ser115, His47 and His131 of the HHV-7 enzyme (Fig. 3, fig004sdr).

A structural representation of the serine protease from HCMV is shown in Figure 4 (fig007sdr), with the catalytic nucleophile (Ser132) coloured in black and the other active site residues coloured purple (note that the enzyme is a homodimer, and that the two monomer subunits are coloured red and green in this figure). More detailed molecular information concerning this enzyme, including a three-dimensional representation of the protein, is available (see mol001sdr, the dynamic molecular model created using CHIME software, which can be viewed using the same software, in the html version only).

Association with disease

The pathogenesis and disease association of HHV-7 have, to date, been poorly described, and much of the information that is currently available is derived from a few case reports that might (or might not) be representative of the typical range of host responses to HHV-7 infection. With this caveat in mind, the pathogenesis and epidemiology of HHV-7 infection are discussed below, and are summarised (along with those of HHV-6) in Table 2 (tab003sdr).

Early, ubiquitous acquisition and persistent shedding in saliva

The majority of children become seropositive for anti-HHV-7 antibodies by the age of three years (Refs 24, 25), and over 95% of adults are found to be seropositive for HHV-7 (Ref. 24). Following primary infection, the virus appears to be shed throughout life in the saliva, and infectious HHV-7

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Table 2. Pathogenesis and epidemiology of human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7) (tab003sdr)

	HHV-6	HHV-7
Paediatric infection	Infects most children at 6–24 months of age (somewhat earlier than HHV-7).	Infects most children by 3 years of age.
Primary infection (infants and children under three years of age)	HHV-6B infection usually manifests as a febrile illness, with or without a rash; presentation with a rash is usually diagnosed as roseola (exanthem subitum). HHV-6A can also cause roseola.	HHV-7 can cause roseola.
Oropharyngeal persistence	DNA can be found in salivary glands and in saliva of a high proportion of adults.	DNA can be found in salivary glands and saliva of a high proportion of adults; infectious virus is readily isolated from saliva of almost all adults.
Neurological involvement (children and adults)	Primary HHV-6B infection in infants is associated with seizures, particularly in the 12–15-month age range, and with some cases of encephalitis. In adults, HHV-6 DNA has been detected in biopsy samples from some cases of focal encephalitis, and viral antigens have been detected in active CNS lesions from persons with multiple sclerosis.	In two Japanese children, primary HHV-7 infection has been associated with exanthem subitum complicated by acute hemiplegia.
Infection in immunocompromised adults	HHV-6A and HHV-6B can be pathogenic; the viruses can cause pneumonitis, bone marrow suppression and encephalitis.	Effects of HHV-7 are uncertain and might be complicated by reactivation of HHV-6.
Role in AIDS	Its role is uncertain, both in adults and in children. *In vitro*, HHV-6 can induce CD4 expression on some $CD4^-$ cells; it can also up-regulate HIV-1 gene expression.	Its role is uncertain, both in adults and in children. *In vitro*, HHV-7 can compete for CD4 binding and inhibit HIV-1 infection.
Other diseases	HHV-6 has been proposed to play a role in CFIDS, but evidence for this is currently equivocal.	Unknown.
	Shared properties of HHV-6 and HHV-7	
Prevalence	Over 90% of all adults are infected with both HHV-6 and HHV-7.	
Reactivation (children)	Children who have previously been infected with HHV-6 can experience reactivation of HHV-6 in conjunction with primary HHV-7 infection; HHV-7 can also reactivate HHV-6 *in vitro*.	

Abbreviations used: AIDS = acquired immunodeficiency syndrome; CFIDS = chronic fatigue immunodeficiency syndrome; CNS = central nervous system; HHV = human herpesvirus; HIV = human immunodeficiency virus.
Other details, including citations, are described in the main text.

can be readily isolated from saliva samples from most adults (Refs 3, 5, 6). Consistent with this, HHV-7 DNA has been detected in salivary glands from a majority of healthy adults (Refs 26, 27, 28).

The persistent, productive state of HHV-7 infection in the salivary glands contrasts with the latent (reversibly non-productive) infection that occurs in PBMCs). Thus, although HHV-7 DNA can be detected in the PBMCs of most healthy adults (Ref. 29), at a level of approximately 300 copies of viral DNA per one million cells (Ref. 28), infectious virus cannot usually be isolated from unstimulated PBMC cultures. HHV-7 can, however, be efficiently isolated from mitogen-stimulated PBMCs (Refs 1, 30), and reactivation of HHV-7 can, in turn, lead to reactivation of HHV-6, from a putative latent state in tissue cultured PBMCs (Ref. 30). The latter observation emphasises the interplay that might occur between HHV-7 and HHV-6, as discussed below.

Primary infection, and association with exanthem subitum (roseola)

In a study published in 1994, Tanaka and co-workers reported that 5/7 infants who exhibited typical signs and symptoms of exanthem subitum underwent seroconversion for HHV-7 immediately following their illness (Ref. 31). Confirmatory reports describing the isolation of HHV-7 from other infants with exanthem subitum were published in 1995 (Refs 32, 33). Nonetheless, the frequency with which HHV-7 can cause roseola or other febrile illness in infants and young children remains unclear. The possible association between HHV-7 infection and more serious complications, such as neurological disease, is also uncertain, although two Japanese children with evidence of primary HHV-7 infection were reported to have had exanthem subitum complicated by seizures and acute hemiplegia, and followed by brain atrophy (Ref. 34).

In a significant number of cases, primary HHV-7 infection has also been reported to be associated with reactivation of HHV-6; this is reflected by rising anti-HHV-6 antibody titres in sera from convalescent infants and/or excretion of HHV-6 DNA in saliva (Refs 33, 35). These findings are consistent with data from *in vitro* experiments, showing that HHV-7 infection of cultured PBMCs can lead to reactivation of latent HHV-6 (see above), as well as data from *in vivo* experiments, showing that HHV-7 and HHV-6 can, at least sometimes, co-infect the same cell (Refs 30, 36). Thus, the pathogenesis of HHV-7 might be inextricably linked with that of HHV-6 (see review article on HHV-6, txt001sdr).

Association with disease in immunocompromised individuals

By analogy to other herpesviruses, and most particularly to HCMV, HHV-7 might be expected to have the potential for pathogenicity in the immunocompromised host. However, this issue has received very little attention, except in the case of HIV-1 infection.

In HIV-1-infected individuals, the frequency of detection and viral load of HHV-7 in saliva have been found to be increased (Ref. 26). It is not clear whether this is, however, clinically significant. In theory, HHV-7 could have a beneficial (suppressive) effect on HIV-1 replication, because the virus can competitively inhibit HIV-1 infection of cultured $CD4^+$ T cells and monocyte-derived macrophages (Refs 8, 11). However, reactivation of HHV-7 infection might also lead to reactivation of HHV-6, and thereby to disease in the immunocompromised host (Refs 4, 30, 33) (see review article on HHV-6, txt001sdr). Further studies will be needed to investigate this issue.

Potential practical applications of HHV-7

The highly selective tropism of HHV-7 for activated $CD4^+$ T cells suggests at least two possible future applications of the virus and its gene products. The first is the development of HHV-7-based viral vector systems, capable either of efficient delivery of exogenous DNA to $CD4^+$ T cells, or of selective replication within such cells. Such vector systems might allow for (1) the introduction of therapeutic genes into HIV-1 target cells (gene therapy), (2) the selective manipulation of T-cell immunity (immunotherapy), and (3) even (perhaps) the eradication of autoreactive $CD4^+$ T-cell clones (immunosuppressive therapy), for the treatment of auto-immune diseases, such as multiple sclerosis.

A second application of HHV-7 might lie in the development of novel pharmacological agents for the suppression of HIV-1 replication. In particular, the putative CD4-binding protein encoded by HHV-7 might serve as a lead compound for the design of novel, high-affinity ligands, which could compete with gp120 of

HIV-1, for binding to CD4. Such competitive, receptor-binding compounds might be particularly powerful if used in combination with other anti-retroviral drugs, including the chemokine-based competitors for the HIV-1 co-receptors.

Research in progress and outstanding research questions

Identification of the HHV-7 gene product(s) that bind to the CD4 receptor

Identification of this ligand would shed more light on the biology of HHV-7, and might also provide a basis for the design of novel drugs that might be able to competitively inhibit HIV-1 infection.

Uncovering the pathogenesis and disease association(s) of HHV-7 infection

It remains unclear whether HHV-7 is associated with neurological diseases (in common with HHV-6), or is associated with other illnesses, either in childhood or in later life. Prospective studies will be needed to address these questions.

Analysis of the effect of HHV-7 infection on HIV-1 replication in vivo

The theoretical negative effect of HHV-7 on HIV-1 replication remains unsubstantiated *in vivo*, but merits investigation, particularly in children with primary HHV-7 infection.

Acknowledgements and funding

Stephen Dewhurst's work has been supported, in part, by US National Institutes of Health grants (RO1 AI34231 and KO4 AI01240). We are grateful to Dr Maurice Zauderer (University of Rochester Medical Center) for helpful discussions. We also thank Drs Mary Caserta (University of Rochester Medical Center), John Nicholas (Johns Hopkins Oncology Center) and Carol Wu (University of Connecticut Health Center) for critically reviewing the manuscript.

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https://doi.org/10.1017/S1462399497000112 Published online by Cambridge University Press

Further reading, other resources and other contacts

A genome database that includes the complete genome of HHV-7
http://ww3.ncbi.nlm.nih.gov/Entrez/Genome/org.html

Tables

Table 1. Genetics and cell infection of human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7) (tab001sdr).

Table 2. Pathogenesis and epidemiology of human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7) (tab003sdr).

Coding capacity of human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7) (tab002sdr, in the html version only).

Schematic figures

Figure 1. Genome organisation of human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7) (fig005sdr).

Figure 2. Schematic diagram of human herpesvirus 6 (HHV-6) *ori*Lyt (fig002sdr).

Figure 3. Alignment of the protein sequences of the serine proteases of human cytomegalovirus (HCMV), human herpesvirus 6 (HHV-6) and human herpesvirus 7 (HHV-7) (fig004sdr).

Figure 4. Structural model of human cytomegalovirus (HCMV) serine protease (fig007sdr).

Other resources

Three-dimensional structure of the serine protease of human cytomegalovirus (HCMV) (mol001sdr, html version only).

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Dewhurst, S., Skrincosky, D. and van Loon, N. (1997) Photograph of a child with exanthem subitum (roseola), Exp. Rev. Mol. Med., fig001sdr, 5 November 1997 (also in review article on human herpesvirus 6, txt001sdr)

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