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Recognition of novel viral sequences that associate with the dynein light chain LC8 identified through a pepscan technique

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Abstract Recent data from multiple laboratories indicate that upon infection, many different families of viruses hijack the dynein motor machinery and become transported in a retrograde manner towards the cell nucleus. In certain cases, one of the dynein light chains, LC8, is involved in this interaction. Using a library of overlapping dodecapeptides synthesized on a cellulose membrane (pepscan technique) we have analyzed the interaction of the dynein light chain LC8 with 17 polypeptides of viral origin. We demonstrate the strong binding of two herpesvirus polypeptides, the human adenovirus protease, vaccinia virus polymerase, human papillomavirus E4 protein, yam mosaic virus polyprotein, human respiratory syncytial virus attachment glycoprotein, human coxsackievirus capsid protein and the product of the AMV179 gene of an insect poxvirus to LC8. Our data corroborate the manipulation of the dynein macromolecular complex of the cell during viral infection and point towards the light chain LC8 as one of the most frequently used targets of virus manipulation.

Key words: Migration; Microtubule; Infection; Dynein; Pepscan

1. Introduction

Dyneins are large protein complexes that function as microtubule-based molecular motors generating force towards the minus end of microtubules, with the intermediate and light chains presumably involved in the dynein attachment to the appropriate cargo [1]. Among the dynein light chains, we have focused our work on the interaction of LC8, an 89-residue protein, with its target sequences. This 8-kDa subunit (LC8 actual mass 10 kDa) was first identified as an integral component of the *Chlamydomonas reinhardtii* outer dynein arm, in association with the dynein intermediate chains at the base of the soluble particle [2]. After the initial cloning and identification of LC8 as a neuronal nitric oxide synthase-interacting protein [3], several other polypeptides were reported to bind to this dynein light chain. The list include the proapoptotic member of the Bcl-2 family Bim [4], the product of the *Drosophila* swallow gene [5], a guanylate kinase domain-associated protein [6], DNA cytosine methyl transferase [7], the transcription factor Kid-1 [7], a microtubule-associated protein [7], dynein heavy chain [7], BS69 [8], the potential oncogene AIBC1 [8], the *Chlamydomonas* radial spoke protein 3 [9] as well as multiple proteins of unknown function [8].

Two different protein motifs can be recognized by LC8 in its target proteins: a (K/R)*X*TQT and a G(I/V)QVD motif [7,8]. Studies using multidimensional nuclear magnetic resonance spectroscopy have revealed that the Gln (Q) present in both motifs occupies an invariant position in the binding cleft of LC8, establishing strong hydrogen bonds with the side chains of Glu35 as well as Lys36 [10]. In both cases, the peptides from the target sequences engage with the LC8 dimer as a novel β-strand that elongates the preformed β-sheet [10,11]. Proteins that interact with LC8 through unknown mechanisms include myosin V [6] and IκBα [12]. Over 20 cellular proteins are currently known to interact with LC8, many of them becoming subsequently transported along microtubules in a retrograde manner. In most cases these target proteins possess one of the two LC8 binding motifs mentioned above.

It is becoming more and more apparent that multiple viruses are able to sequester the dynein machinery of the infected cell in order to move along the microtubules and gain access to the cell nucleus [13–15]. In the case of both rabies virus and Mokola virus (lyssavirus), the yeast two-hybrid technology has revealed a direct interaction between the KSTQT motif present in the viral P protein and LC8 [16,17]. Likewise, African swine fever virus (Asfarviridae) has been reported to interact with LC8 through the TASQT motif present in the viral polypeptide p54 [18]. We have synthesized overlapping dodecapeptides covering the sequences of viral proteins corresponding to papillomavirus, retrovirus (HIV and EIAV), togavirus, poxvirus (entomopoxvirus, vaccinia and variola virus), potyvirus (yam mosaic potyvirus), paramyxovirus (respiratory syncytial virus), adenovirus, herpesvirus, rotavirus, and coxsackievirus. Positive interactions between LC8 and several viral proteins have been observed, reinforcing the idea that the utilization of the dynein motor could be a broad strategy adopted by a wide variety of viruses of unrelated families.

2. Materials and methods

2.1. Peptide synthesis

Overlapping dodecapeptides for mapping studies were prepared by automated spot synthesis (Abimed, Langenfeld, Germany) onto an amino-derivatized cellulose membrane, immobilized by their C-termi-

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ni via a polyethylene glycol spacer, and N-terminally acetylated. We used a sequential displacement of two amino acids during the synthesis, which leads to an overlap of 10 residues between each two consecutive dodecapeptides, an 8-residue overlap among three consecutive dodecapeptides, a 6-residue overlap among four consecutive dodecapeptides, and so on [7].

2.2. Recombinant LC8 binding assays on cellulose-bound peptides

Purification of recombinant LC8 has been described previously [19]. The cellulose membranes were coated with 1% non-fat dried milk in TBS (50 mM Tris, pH 7.0, 137 mM NaCl, 2.7 mM KCl) for 4 h at room temperature. Incubation with the recombinant LC8 (0.13 μM) was done overnight at room temperature [7]. Subsequently, the membrane was incubated for 2 h at room temperature with a commercial antibody against the hexahistidine tag present in the recombinant protein (1:100 000 dilution in TBS). Development of the membrane was performed by ECL following the manufacturer's instructions. The quantification of the intensity of each spot was performed utilizing a UVI-tec digital image analyzer (UVItec, Cambridge, UK) and the software UVIband V97. In every case, spots corresponding to the dodecapeptides of the various proteins synthesized onto the same membrane were compared with each other. Controls performed with the antibody in the absence of recombinant LC8 were performed in every case, in order to subtract the non-specific binding due to the reactivity of the antibody against certain synthetic dodecapeptides.

3. Results

Considering the existence of consensus LC8 binding motifs in certain viral polypeptides that might result in a retrograde transport along microtubules, we performed a homology search within viral sequence databases. When putative LC8 binding motifs or homologous sequences were found, we synthesized large portions of the selected viral protein as overlapping dodecapeptides on a cellulose membrane that was subsequently incubated with recombinant LC8. We focused initially on herpes- and adenovirus, since these two virus families are known to interact with the dynein motor.

3.1. Binding of LC8 to herpesvirus proteins

Two putative LC8-interacting proteins were selected: the herpes simplex virus type 1 helicase (product of the U_L9 gene) as well as the product of the human herpesvirus 6B U19 gene. The human helicase possesses a KSTQT motif whereas the product of the U19 gene possesses two contiguous RSTQT motifs (see arrows in Fig. 1). This KSTQT motif is also found in several LC8-interacting proteins, such as Bim or Kid-1 [7,8], whereas the RSTQT motif includes a conservative R substitution. One hundred and sixty amino acids of each protein were synthesized as overlapping dodecapeptides that were subsequently screened using the pepscan technique. When recombinant LC8 was incubated with 75 overlapping synthetic dodecapeptides of each protein, we could observe a positive interaction in both cases (Fig. 1).

3.2. Binding of LC8 to adenovirus protease and to BS69

After a search for putative LC8-interacting domains, we tested the binding of recombinant LC8 to the human adenovirus protease as well as to BS69, a cellular protein with transactivating properties that binds tightly to adenovirus E1A using the pepscan technique (Fig. 2). A weak but reproducible binding was observed in a KSTQT motif present in the adenoviral protease, as well as a strong binding to VSTQT and HRSTQT motifs present in BS69. The lower signal observed in the case of the KSTQT motif present in the adenovirus protease could be due to the two Val residues that flank the

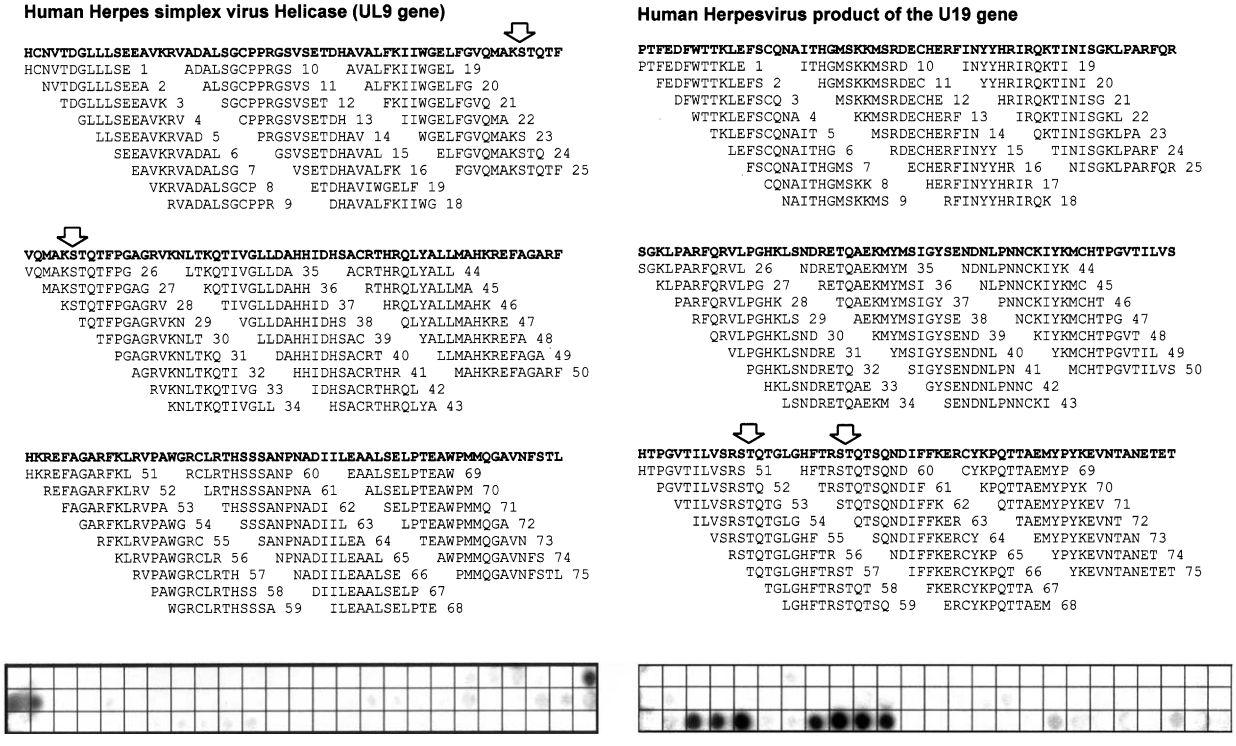

Fig. 1. Binding of LC8 to herpesvirus helicase and to the product of the U19 gene. Recombinant LC8 was incubated with a cellulose membrane bearing 75 spots with synthetic dodecapeptides that cover 160 amino acids of each protein. Twenty-five dodecapeptides were synthesized on each line. The membrane was developed by ECL after incubation with a peroxidase-labeled anti-hexahistidine tag antibody. The arrows mark the protein sequences that interact with LC8 (KSTQT in the helicase and RSTQT in the product of the U19 gene).

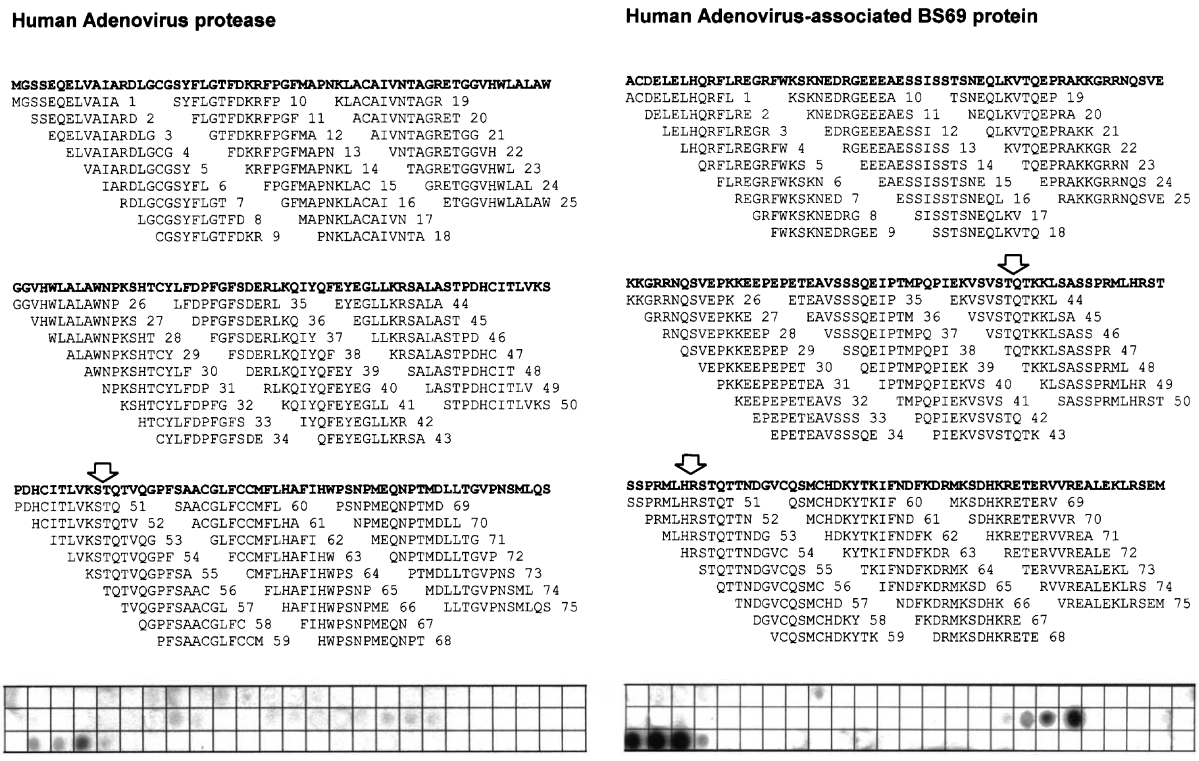

Fig. 2. Binding of LC8 to adenovirus protease and to the human adenovirus-associated BS69 protein. Recombinant LC8 was incubated with a cellulose membrane bearing 75 spots with synthetic dodecapeptides that cover 160 amino acids of each protein. Twenty-five dodecapeptides were synthesized on each line. The membrane was developed by ECL after incubation with a peroxidase-labeled anti-hexahistidine tag antibody. The arrows mark the protein sequences that interact with LC8 (KSTQT in the protease and VSTQT and RSTQT in the BS69 protein.

LC8 consensus binding motif [7]. Hence, it is likely that the three-dimensional fold adopted by the full-length protease in solution promotes its interaction with LC8.

3.3. Binding of LC8 to other viral targets

Even if a viral polypeptide possesses a consensus LC8 binding motif such as KSTQT or GIQVD, it is important to inspect the neighboring residues, since they determine if the interaction will eventually take place. On the other hand, some variability can still be observed at the recognition sequences [7,8]. Hence, we synthesized dodecapeptides covering 14 viral proteins that resembled the consensus LC8 binding

Table 1
Analyzed protein sequences that failed to bind to LC8

Variola virus CR6 protein (7514341)
WAYLSKKDTGIEFVDNDRQDIYTLFQHTGRIVHSNLTETFRDYIFPGDKTSYWVWLNESI

Variola virus DR4 (9627525)
WKKRIAGRDYMTNLSRDTGIQQSNLTETIRNCQKNRNIYGLYIHYNLVINVVIDWITDVI

Amsacta moorei entomopoxvirus spheroidin (9964501)
RYGEFEVYNADTGLIYAKNLSIKN*Y*DTVIQVERLPVNLKVRAYTKDENGRNLCLMKITSS

HIV matrix protein (7715890)
LERFAINPGLLETSEGCQQIIEQLQSTLKTGSEELKSLFNTVATLWCVHQRIEVKDTREA

EIAV core protein (13383732)
MVSIAFYGGIPGGISTPITQQTKSTDTQKGDHMVYQPYCYNDSHKAEMAEARDTRYQEEM

Rubella virus C structural protein (16923735)
RRRGNRGRGQRRDWSRAPPPPEERQESRSQTPAPKPSRAPPQQPQPPRMQTGRGGSAPRP

Human poliovirus 1 VP2 protein (1335516)
SPNIEACGYSDRVLQLTLGNSTITTQEAANSVVAYGRWPEYLRDSEANPVDQPTEPDVAA

Rotavirus VP4 protein (1519243)
LKEMATQTDGMNFDDISAAVLKTKIDKSTQLNTNTLPEIVTEASEKFIPNRAYRVIKDDE

The accession numbers are shown in parentheses and the sequence motifs initially suspected to bind to LC8 are underlined. In several cases larger stretches of sequence were analyzed, although only 60 residues are shown for clarity.

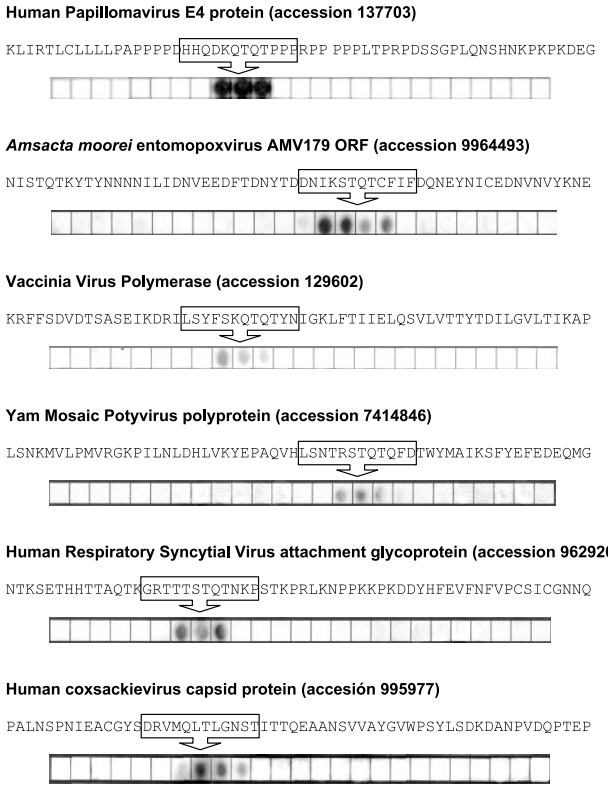

Fig. 3. Binding of LC8 to additional viral targets. Recombinant LC8 was incubated with a cellulose membrane bearing spots with synthetic dodecapeptides that cover sequences corresponding to six different viral proteins. For clarity, only 25 dodecapeptides (60 amino acids) are shown on each line. The membrane was developed by ECL after incubation with a peroxidase-labeled anti-hexahistidine tag antibody. The arrows and boxes mark the protein sequences that interact with LC8 together with the dodecapeptide that showed the strongest binding to LC8. Accession numbers are shown in parentheses.

motifs and incubated them in the presence of recombinant LC8. Special care was taken when searching for homology within sequences present in polypeptides of virus known to bind to dynein or to require intact microtubules for effective infection. Additionally, we also inspected several virus sequences that were recently proposed to bind to LC8 according to homology studies [20]. In all cases, we screened a minimum of 60 residues (25 spots) and a maximum of 110 residues (50 spots).

A very strong interaction was observed in the case of human papillomavirus E4 protein, *Amsacta moorei* entomopoxvirus, vaccinia virus polymerase, yam mosaic virus polyprotein, human syncytial virus attachment glycoprotein and human coxsackievirus A3 capsid protein (Fig. 3). In all cases, LC8 binding was observed to sequences that include a Gln residue (Q). In five out of the six viral sequences that interacted with LC8, the Gln residue was followed by a Thr residue. Conversely, eight other viral sequences failed to interact with LC8, despite their sequence similarity with the LC8 binding sequences present in target proteins (Table 1).

4. Discussion

The absolute requirement of intact microtubules for efficient herpesvirus infection as well as the interaction of the virus with the dynein machinery is well established (see [21] and references therein). In epithelial cells and in axons, incoming cytosolic capsids are transported along microtubules towards the nucleus [22,23]. In fact, the herpes simplex virus U_L34 protein has been reported to interact with the neuronal isoform of the intermediate chain of cytoplasmic dynein [24], although this polypeptide is not present in secreted virions. Remarkably, one of the polypeptides that interacted with LC8 (Fig. 1) was the product of the herpes simplex 1 U_L9 gene, a protein with helicase and DNA binding activities [25]. It remains to be established if, after viral uncoating, the retrograde transport of the helicase towards the nucleus is indispensable for the viral life cycle. Additionally, a very strong interaction was observed between LC8 and the product of the human herpesvirus U19 gene, a protein of 325 amino acids of unknown function (not to be confused with the product of the UL19 (VP5) gene of HSV-1, the major capsid protein) [26]. Two contiguous binding sites were observed in these polypeptides, both of them displaying consensus LC8 recognition sequences (Fig. 1).

As in the case of herpesvirus, numerous reports have described the microtubule-dependent retrograde transport of adenovirus during infection [23,27–29]. In fact, injection of function-blocking antibodies against cytoplasmic dynein, but not kinesin, blocked nuclear localization of adenoviruses, consistent with a minus-end-directed motility [29]. Using the pepscan technique, we could also identify an interaction between human adenovirus protease and recombinant LC8 (Fig. 2). Although the consensus KSTQT is present in the adenoviral protease, the two flanking Val residues probably contribute to the reduction of the binding affinity, as previously shown when this motif was inserted in a poly-Val dodecapeptide [7]. Confirmation of this interaction would be tentatively obtained with co-immunoprecipitation or co-immunofluorescence studies using anti-protease antibodies. In addition, we also screened human adenovirus E1A-associated BS69 protein for LC8-interacting sequences. Using LC8 as bait in a yeast two-hybrid experiment, this cellular protein was equally fished using a rat cDNA library [8]. BS69 is a transcriptional corepressor protein and a potential tumor suppressor that binds to the adenoviral oncoprotein E1A [30]. Incubation of recombinant LC8 with BS69 dodecapeptides results in two positive interactions: a weak binding to VSTQT and a very strong binding to the RSTQT motif (Fig. 2). As previously mentioned, adenovirus type 2 E3 14.7-kDa protein also interacts with the dynein light chain TCTEL1 using a small

Table 2
Summary of viral sequences that interact with the dynein light chain LC8

Rabies virus P protein	KSTQT
Mokola virus P protein	KSTQT
African swine fever virus p54	TASQT
Human herpes simplex helicase	KSTQT
Human herpesvirus U19 gene	RSTQT
Human adenovirus protease	KSTQT
Human papillomavirus E4 protein	KQTQT
A. moorei entomopoxvirus AMV179 ORF	KSTQT
Vaccinia virus polymerase	KQTQT
Yam mosaic potyvirus polyprotein	RSTQT
RSV attachment glycoprotein	TSTQT
Human coxsackievirus capsid protein	RVMQL

The interaction between the first three proteins and LC8 has been reported in [7,16–18].

GTPase as a bridge protein [28]. Thus, it is likely that the full picture of adenovirus–dynein interactions might involve additional proteins not yet identified.

Six additional polypeptides interact with LC8 according to our pepscan analysis, many of them belonging to viruses known to associate with microtubules (Fig. 3). We observed that papillomavirus E4 protein uses a KQTQT motif in order to interact with LC8, in agreement with previous observations reporting that nocodazole treatment of infected cells does not completely impair internalization of bovine papillomavirus but retains the incoming virions in cytoplasmic vesicles, hence abrogating their transport towards the nucleus [31]. Our finding that vaccinia virus polymerase interacts (albeit weakly) with LC8 through a KQTQT motif is also noteworthy. The requirement of dynein integrity for vaccinia virus infection has been demonstrated through different approaches. Disruption of the microtubular network of infected cells with nocodazole altered the localization of vaccinia virus at the microtubule organizing center (MTOC), and infected cells overexpressing p50/dynamitin which acts as a dominant-negative for dynein–dynactin function, abrogated the correct positioning of the virus during infection [32]. Further experiments will be necessary in order to shed light on the strong interaction that we observed between the human RSV attachment glycoprotein and LC8 (Fig. 3), since this protein is located in the extracellular domain of the virus.

Despite the recently published interaction of retrovirus with the dynein machinery [33], we have been unable to obtain binding of LC8 to HIV matrix protein or to identify any LC8 consensus binding sequence within the HIV genome. A putative LC8 binding motif was nevertheless observed in the core protein of the retrovirus EIAV (KSTDTQ), although incubation with recombinant LC8 rendered no interaction (Table 1). The most likely explanation to the reported dynein-dependent retrograde transport of HIV might be that one of the polypeptides present in this retrovirus must interact either with dynactin, with another dynein light chain or intermediate chain or via a bridging protein, as reported in the case of adenovirus E3 protein.

It must be noted as well that rotavirus P4 protein and poliovirus 1 VP2 protein, two of the polypeptides that Poisson and coworkers proposed to bind to LC8 [20], failed to do so according to our assay (Table 1). This observation suggests that notwithstanding the plasticity of the binding cavity within LC8 which is able to adapt to a considerable number of peptide variants of the two consensus motifs, it is difficult a priori to predict a positive interaction. Additionally, the interactions established between the residues present in the viral polypeptides that flank the consensus LC8 binding motifs must be determinant for a successful interaction to take place.

Significantly, it has been shown that while the LC8 binding motif in the P protein of rabies virus is important for binding to LC8, it does not affect neurotropism unless it is disrupted in an already attenuated virus mutant [34]. In fact, LC8 co-purifies with wild-type virions of rabies virus isolated in a sucrose gradient, but not with virions containing phosphoprotein mutants with alterations in the KSTQT motif [34].

The approach that we describe in this work is complementary to the more common yeast two-hybrid screening. Moreover, with the pepscan technique we can inspect if a large variety of viral sequences interact with LC8, whereas no viral libraries containing multiple, unrelated, virus genes are commercially available. The list of plant and animal viruses known to associate with the microtubule cytoskeleton or directly with microtubules in vitro is significant [13–15]. Indeed, there is a growing list of viruses, at least those requiring nuclear import for replication, that use dynein to approach the perinuclear position of the MTOC. It is also widely recognized that virus infection often resulted in disruption of the cytoskeleton. Our results suggest that the genome of as many as 12 viruses belonging to distant families have evolved introducing LC8 recognition sequences that might allow these viruses to associate with the dynein motor and become transported towards the minus end of microtubules after infection. Interestingly, in all cases, modifications of the KSTQT motif rather than the GIQVDR motif are observed (Table 2). However, further experiments will be necessary in order to demonstrate that the proteins described in this work that interact with LC8 using the pepscan technique are able to recruit the complete dynein motor in cells. In addition, since LC8 is also a subunit of myosin V, the possibility exists that, at least in certain cases, the interaction of LC8 with certain viral proteins results in plus-end-directed microtubular transport. In summary, our data provide support for the indispensable role of the macromolecular motor dynein in viral infection and identify novel protein–protein interactions endowed with physiological relevance.

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