Interaction of the Poliovirus Receptor CD155 with the Dynein Light Chain Tctex-1 and Its Implication for Poliovirus Pathogenesis*

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Steffen Mueller‡, Xuemei Cao§, Reinhold Welker, and Eckard Wimmer¶
From the Department of Molecular Genetics and Microbiology, State University of New York, Stony Brook, New York 11794

The cellular receptor for poliovirus CD155 (or PVR) is the founding member of a new class of membrane-associated immunoglobulin-like proteins, which include the mouse tumor-associated antigen E4 (Tag4) and three proteins termed “nectins.” Using a yeast two-hybrid screen we have discovered that the cytoplasmic domain of CD155 associates strongly and specifically with Tctex-1, a light chain of the dynein motor complex, the latter representing the major driving force for retrograde transport of endocytic vesicles and membranous organelles. We confirmed the interaction biochemically and by co-immunoprecipitation, and we mapped the Tctex-1 binding site to a SKCSR motif within the juxtamembrane region of CD155. Tctex-1 immunoreactivity was detected in mouse sciatic nerve and spinal cord (two tissues of central importance for poliovirus pathogenesis) in punctate, possibly vesicular, patterns. We propose that the cytoplasmic domain may target CD155-containing endocytic vesicles to the microtubular network.

Neurotropic viruses like poliovirus, herpesvirus, rabies virus, and pseudorabies virus all utilize neuronal retrograde transport to invade the central nervous system. Association with Tctex-1 and, hence, with the dynein motor complex may offer an explanation for how poliovirus hijacks the cellular transport machinery to retrogradely ascend along the axon to the neuronal cell body.

Poliovirus, a member of the genus Enterovirus of Picornaviridae, is a human neurotropic virus that causes poliomyelitis, a disease leading to paralysis or death. Although discovered nearly one hundred years ago, the mechanism by which poliovirus invades the central nervous system (CNS) and specifically selects motor neurons for destruction remains elusive.

Paralytic poliomyelitis is a rare neurologic complication of poliovirus infection (one out of one hundred), as the virus enters humans by ingestion and proliferates largely unnoticed in the gastrointestinal tract. Interestingly, once in the CNS, the virus spares most cells of the CNS parenchyma other than motor neurons from destruction. This unique neurotropism has attracted attention for decades, but no satisfactory explanation has been presented.

A major determinant of a virus’ pathogenicity is its tissue tropism, which is governed predominantly by the cognate cell surface receptor. The receptor for all three serotypes of poliovirus, CD155 (also known as PVR), is a membrane-associated, single-span glycoprotein belonging to the immunoglobulin superfamily (1). The CD155 gene expresses four splice variants of which two are membrane-bound (CD155α and β) and function as poliovirus receptors. CD155β and γ are secreted isoforms lacking a transmembrane domain (2).

Impaired by the apparently exclusive restriction to primates, CD155 is only slowly revealing its physiological functions. CD155 mRNA expression has been detected by Northern blot analyses in brain, small intestine, placenta, heart, skeletal muscle, kidney, lung, and liver (3, 4). Similar albeit not identical expression patterns have been found in transgenic mice expressing the human CD155 gene (5, 6). CD155-tg mice are now well established as a model of human poliomyelitis. Activity of the CD155 promoter in the notochord and floor plate of the developing neural tube of CD155 promoter/LacZ-tg mice suggests that a primary function of CD155 may relate to the development of the CNS during embryogenesis (7).

The sites of primary infection and gastroenteric replication of poliovirus are thought to be in tonsillopharyngeal tissue and the Peyer’s patches of the small intestine (8, 9). The commonality between these structures is their large concentration of lymphatic cells, but, remarkably, the predominant cells supporting poliovirus proliferation in the gastrointestinal tract of an infected individual remain unknown.

As aforementioned, poliovirus infections are mainly restricted to the enteric tract. Viremia, however, is a prerequisite for the progression to poliomyelitis. How poliovirus spreads subsequently from the blood to the CNS is poorly understood. Two pathways have been suggested. First, the virus may directly pass from the blood into the CNS by crossing the blood-brain barrier (10). The second hypothesis suggests that the virus is transported by retrograde axonal transport ascending from the muscle to the spinal cord and brain (11). This hypothesis has found strong support in experiments with CD155-tg mice (12–14). The presence of poliovirions in axons during poliomyelitis has been a long observed phenomenon (15, 16). Ohka and colleagues (12) provided direct evidence of poliovirus in the sciatic nerves of tg mice and showed that during retrograde axonal transport the virus remains in the form of an intact 160 S particle.
In this study we have focused on one or more possible functions of the intracellular C-terminal domain of CD155 to decipher cell-internal interactions important for protein function, perhaps related to poliovirus pathogenesis. We have therefore analyzed the C-terminal peptides of CD155α and -δ in a yeast two-hybrid screen. Here we describe the interaction of the cytoplasmic domain of CD155 with Tctex-1, a light chain subunit of the dynein motor complex. Considering the function of dynein as the major driving force for minus end-directed transport along microtubuli, we propose that we have identified a driving force for cytoplasmic transport machinery. Indeed, we consider it possible that the majority of poliovirus infection of the CNS leading to destruction of motor neurons may occur via retrograde axonal transport.

We discuss our results in light of most recent findings for the transport machinery. Indeed, we consider it possible that the dynein as the major driving force for minus end-directed transcytoplasmic domain of CD155 with Tctex-1, a light chain subdomain was inserted into pGEX-KG (22). A mutant pGEX-pCDNA-Tctex-1 using the coupled wheat germ in vitro transcription/translation system (TntB from Promega) according to the manufacturer’s instructions. 100 μg of GST fusion protein was loaded to beads, which was incubated with 20 μl of translation product for 30 min at 30 °C, agitating the mixture every 2–3 min, followed by four washes with binding buffer.

Bound proteins were separated by SDS-PAGE gel and visualized by autoradiography.

In a second type of GST pull-down experiment, a cytoplasmic cell lysate of the mouse neuroblastoma cell line Neuro2A was used as a source of endogenous Tctex-1. Neuro2A cells were lysed in cold lysis buffer (10 mM Tris, pH 7.4, 150 mM NaCl, 1 mM EDTA, 1% Triton X-100, 0.5% Nonidet P-40, 0.1% deoxycholate, 10% glycerol) containing protease inhibitors. An equivalent of 5 × 10^6 cells was incubated with 50 μg of GST fusion protein on beads for 3 h at 4 °C under agitation. Beads were washed three times with lysis buffer, followed by SDS-PAGE of the eluted proteins and Western blotting with rabbit polyclonal antibody R5205 against Tctex-1.

In a third pull-down strategy we combined the efficacy of the GST system with the convenience of detection of AP fusion proteins. Plasmid pCDNA-AP-CD155α-seap expression plasmid pAPtag4 were transfected into HEK 293 cells. One milliliter of conditioned medium (containing 1500 μCi/ml of AP fusion proteins) was incubated with 100 μg of GST fusion proteins bound to beads and agitated for 2 h at room temperature. The beads were then washed four times with PBS, and the amount of AP protein that was bound to the beads was determined using 9 mg/ml p-nitrophenyl phosphate (in 1 M diethanolamine, pH 9.8, 0.5 mM MgCl2) as a colorimetric substrate.

**Immunological Methods**

**Western Blotting**—For analysis of Tctex-1 protein expression, lysates of mouse tissues from adult ICR mice were prepared by sonication. Equal amounts of protein were separated by SDS-PAGE and blotted onto nitrocellulose membranes (Schleicher & Schuell) followed by Western blot analysis with anti-Tctex-1 antibodies. Intensity of Tctex-1-immunoreactive bands on the exposed film was determined by pixel count analysis of the scanned image with IMAGE 1.61 software (National Institutes of Health).

**Co-immunoprecipitation**—HEK 293T cells were co-transfected with CaP1, a precipitates of plasmids pCDNA-Tctex-1 and pCDNA-pCDNA-CD155(α/δ). Three days following transfection, cells were lysed and processed as described above for Neuro2A cells. The lysate of 5 × 10^6 cells was incubated with anti-dynein (IC74) antibody 70.1, a control IgG antibody (anti-mNectin-2, clone 6B3), or anti-CD155 antibodies (D171 and P44). Immune complexes were captured by the addition of Protein A/G Plus-agarose beads (Santa Cruz Biotechnologies). The precipitated proteins were analyzed by Western blotting using anti-Tctex-1 antibodies.

**Immunofluorescence**—Fresh, unfixed mouse lumbar spinal cord and sciatic nerve tissues were frozen in O.C.T. cryoprotectant (Tissue-Tek). E13.5 mouse embryos were fixed for 2 h in 4% paraformaldehyde before cryoprotection. Frozen sections were collected on poly-l-lysine-coated slides, air-dried, and fixed in cold 1% methanol/acetic for 90 min at -20 °C. Sections were blocked with PBS containing 5% normal horse and 2% normal goat serum for 2 h at room temperature, followed by overnight incubation at 4 °C with anti-Tctex-1 antibodies (17) at a dilution of 1:200 in blocking buffer. After 2 h of washing with PBS, the sections were incubated with a 1:500 dilution of Cy3-conjugated anti-rabbit antibody (Jackson ImmunoResearch) for 1 h at 37 °C. The sections were washed for 2 h in PBS and mounted with Immu-Mount.
Along their respective ORFs. While this study was ongoing, we share complex testis expressed-1) gene (28). CW-1 and mouse Tctex-1...watered at 55 °C for 16 h with 32P-labeled probes, made from a DNA fragment containing the complete CW-1 (Tctex-1) sequence (nucleotides 1–713) by the random primed DNA labeling system (Invitrogen).

RESULTS

Isolation of CD155-binding Proteins—To identify cytoplasmic proteins that interact with CD155, the intracellular domains of CD155α and CD155β were used as baits in a yeast two-hybrid screening procedure similar to that originally described (26, 27). The majority of His+ colonies showed LacZ activity, expressing different levels of β-galactosidase, as assessed by filter binding assay (data not shown). Four classes of strongly to weakly lacZ-expressing clones were isolated. One group of most strongly positive cDNA clones, termed pCW-1, was isolated from yeast and co-transformed with the plasmid pLexA-CD155α or pLexA-CD155β. pCW-1 conferred resistance to His+ selection only when co-transformed with pLexA-CD155α or pLexA-CD155β but not with a control plasmid, pLexA-lam (Fig. 2).

As shown in Table I, β-galactosidase activity was detected only in yeast in which plasmids pCW-1 and pLexA-CD155α or pLexA-CD155β were co-expressed. There was no activity with either Gal4 or LexA-expressing plasmids, both plasmids together, or when co-transformed with pCW-1 and pLexA-lam. About 2000 units of β-galactosidase activity were produced when the cytoplasmic domains of CD155 and full-length CW1 (Tctex-1, see below) were provided (Table I). This indicated that there is a strong interaction between the cytoplasmic domains of either CD155α or CD155β and the CW-1 fusion protein expressed in this yeast two-hybrid system.

CW-1 Is the Human Homolog to the tctex-1 Gene of the Mouse t Complex, a Subunit of Dynein—Sequence analysis of the clone pCW-1 revealed a fragment of ~710 bp fused to the Gal4 transcriptional activation domain. The predicted open reading frame (ORF) was 339 nucleotides long. DNA sequences of 10 individual cDNA clones of pCW-1 were analyzed. All cDNA clones corresponded to the same gene (our GenBankTM accession number U56255), which upon BLAST search was found to be highly homologous to the product of the mouse tctex-1 (t complex testis expressed-1) gene (28). CW-1 and mouse Tctex-1 share ~88% nucleotide identity and ~94% amino acid identity along their respective ORFs. While this study was ongoing, Watanabe et al. (29) also cloned the human homolog of mouse Tctex-1, identical to our sequence, and named it TCTEL1. We will continue to use the original designation Tctex-1 for the human protein throughout this report.

Subsequently, Tctex-1 was identified as a light-chain subunit of the dynein motor complex (18, 30). Dynein exists in two isoforms. Cytoplasmic dynein represents the major driving force of retrograde transport of membrane-bound organelles and vesicles and is involved in spindle movement during mitosis. Axonemal dynein, on the other hand, is the force generator responsible for flagellar and ciliary beating, as is observed in the flagella of sperm or the cilia of the respiratory tract epithelium. Tctex-1 was found to be present in both forms of dynein (18, 30).

Tctex-1 Expression in Mouse and Human Tissues—Except for the overexpression of Tctex-1 mRNA in t-haplotype mice (28), little information is available regarding the expression profile of Tctex-1, particularly in humans. We, therefore, performed Northern blot analysis to determine the expression of the Tctex-1 mRNA in human tissues. A radiolabeled DNA probe corresponding to the entire Tctex-1 cDNA detected a single 0.75- to 0.8-kb band in all tissues tested (Fig. 3A). The highest expression of Tctex-1 was observed in skeletal muscle and testis (Fig. 3A, lanes 6 and 12), whereas the lowest expression was seen in brain and thymus (lanes 2 and 10). This expression pattern matches that observed in mouse and rat (28, 31).

Because CD155-tg mice are a widely used model for the study of poliomyelitis, we were interested in whether tissue-specific patterns in the expression level of Tctex-1 protein reveal similarities to the patterns observed by Northern blot analyses. For this purpose we prepared whole tissue lysates (see “Experimental Procedures”) and analyzed mouse Tctex-1 protein expression by Western blotting. Tctex-1 protein could be detected in all tissues tested, albeit at differing amounts. Protein levels in mouse tissues largely agreed with the mRNA expression in humans (Fig. 3). Especially the high expression in testis (Fig. 3B, lane 7) and low expression in CNS (lanes 1–3) and liver (lane 6) confirm previously reported results (28, 31). However, the very high levels of Tctex-1 mRNA expression in skeletal muscle in humans is in disagreement with the low levels of Tctex-1 protein found in mouse skeletal muscle (compare Fig. 3A, lane 6, and Fig. 3B, lane 4). The reason for this discrepancy is not known. In several tissues the anti-Tctex-1 antibodies recognized a doublet of bands. The presence of a second band was most evident in uterus, spleen, and kidney (Fig. 3B, lanes 9–11). Mou and coworkers (32) found Tctex-1 to be phosphorylated by the Src family kinase p59v 

| DNA-binding domain hybrid | Activation domain hybrid | β-Galactosidase activity |
|---------------------------|-------------------------|------------------------|
| LexA                      | Gal4                    | <1                     |
| LexA                      | GalTctex-1              | <1                     |
| LexA-lam                  | Gal4-Tctex-1            | <1                     |
| LexA-CD155α-cyt           | Gal4-Tctex-1            | 2.38 × 10^3            |
| LexA-CD155β-cyt           | Gal4-Tctex-1            | 2.48 × 10^3            |

a) No plasmid was co-transformed.
CD155 Interaction with Dynein

affinity-purified rabbit polyclonal antibody (17) in combination with a Cy3-conjugated anti-rabbit secondary antibody, we detected Tctex-1 protein in both spinal cord and sciatic nerve (Figs. 4, A, and C). Tctex-1 immune reactivity was higher in embryonal spinal cord and dorsal root ganglia (E13.5; Figs. 4E, A, and C). A weak staining was noted in adult spinal cord and sciatic nerve (Fig. 4A and C). In a first set of experiments, the GST fusion proteins, bound to glutathione-Sepharose beads, were then used in a GST pull-down assay (36) to determine affinities to Tctex-1 protein under the conditions of the assay (Fig. 6, lane 2, 100 μg of total protein loaded per lane).

Analysis of the CD155-Tctex-1 Interaction in Vitro—To confirm the interaction of CD155 with Tctex-1, we generated and expressed in E. coli fusion proteins between glutathione S-transferase (GST) and Tctex-1 or the intracellular C-terminal portion of CD155a (GST-CD155α-cyt, Fig. 6A). In a first set of experiments, the GST fusion proteins, bound to glutathione-Sepharose beads, were then used in a GST pull-down assay (36) to determine affinities to in vitro translated [35S]-Tctex-1. GST-CD155α-cyt specifically bound Tctex-1 (Fig. 6B, lane 4), whereas GST alone did not interact with Tctex-1 (lane 2). We also noticed a weak homo-interaction of Tctex-1 with GST-Tctex-1 (lane 3), an observation supporting a recent report by DiBella and co-workers (33) who demonstrated that Tctex-1 may form a homodimer within the dynein complex.

To ascertain whether CD155α could also interact with endogenous Tctex-1 present in neuronal cells, we extended the GST pull-down experiments and replaced Tctex-1 radiolabeled by in vitro translation with Tctex-1 from cytoplasmic lysates of mouse Neuro2A cells (Fig. 6C). The material bound to the glutathione beads was analyzed for the presence of Tctex-1 immunoprecipitated by Tctex-1. Neither glutathione beads alone, nor GST complexed to glutathione-Sepharose beads interacted with cellular Tctex-1 under the conditions of the assay (lanes 2 and 3, respectively). Because both membrane-bound splice variants of CD155, CD155α, and CD155β, interacted with Tctex-1 in the two-hybrid system, we suspected that the target sequence for Tctex-1 binding lay within the 17 N-terminal cytoplasmic residues that both splice variants have in common (see Fig. 1B). Mok and colleagues (37) have proposed the sequence (R/K)(R/K)XXK(R/K) as a Tctex-1 consensus recognition motif. The N-terminal portions of both CD155 cytoplasmic domains contain the sequence SKCSR, which partly matches this motif (Fig. 1B). We therefore produced a CD155α mutant protein (CD155α-cm) in which the SKCSR sequence was changed to SXXSK(R/K)(R/K). Mok and colleagues (37) have proposed the sequence (R/K)(R/K)XXK(R/K) as a Tctex-1 consensus recognition motif. The N-terminal portions of both CD155 cytoplasmic domains contain the sequence SKCSR, which partly matches this motif (Fig. 1B). We therefore produced a CD155α mutant protein (CD155α-cm) in which the SKCSR sequence was changed to SXXSK(R/K)(R/K). Mok and colleagues (37) have proposed the sequence (R/K)(R/K)XXK(R/K) as a Tctex-1 consensus recognition motif. The N-terminal portions of both CD155 cytoplasmic domains contain the sequence SKCSR, which partly matches this motif (Fig. 1B).
sequence, indeed, is critical for the interaction between CD155 and Tctex-1.

We then developed a different assay in which we combined the GST system with the ease of detection of alkaline phosphatase-tagged proteins. The cytoplasmic domain of CD155a was fused to the C terminus of human placental secreted alkaline phosphatase (AP-CD155α-cyt, Fig. 7A). AP-CD155α-cyt fusion protein and untagged AP protein were expressed in HEK 293 cells from which they were secreted into the culture medium of the cells. After 5 days of expression the medium contained 1500 A405 nm/ml/h of alkaline phosphatase activity, as assessed by a colorimetric assay using p-nitrophenyl phosphate as a substrate for alkaline phosphatase (see “Experimental Procedures”). To confirm the integrity of the secreted proteins, we immunoprecipitated the AP proteins from the supernatant with an alkaline phosphatase-specific antibody and analyzed the immune complexes by Western blot using the same antibody (Fig. 7B). As expected, AP-CD155α-cyt appeared to be slightly larger than AP, due to the addition of 50 amino acids comprising the cytoplasmic domain of CD155α (Fig. 7B, lane 2). The secreted AP and AP-CD155α-cyt were then incubated with glutathione beads to which GST fusion proteins were bound. Although none of the GST fusion proteins bound any significant amount of untagged AP protein (Fig. 7, bars 2, 4, 6), GST-Tctex-1-loaded beads specifically captured AP-CD155α-cyt from the medium (bar 1). No interaction was seen between AP-CD155α-cyt and GST alone (bar 5). Finally, under the conditions of the assay, there was also no homo-interaction observed between CD155 cytoplasmic tails, because we detected...
CD155 Interaction with Dynein

no signal after incubation of AP-CD155α-cyt with GST-CD155α-cyt (bar 3).

CD155 and Tctex-1 Interact in Vivo—To test whether an association between CD155 and Tctex-1 could be observed in tissue culture cells, we performed co-immunoprecipitation experiments. HEK 293T cells were co-transfected with expression plasmids for CD155 and Tctex-1. Cell lysates were subjected to immunoprecipitation using anti-dynein IC74 mAb 70.1 (Fig. 8, lane 2), an unrelated IgG control antibody (lane 3), or a mix of monoclonal anti-CD155 antibodies D171 and P44 (lane 4). Immune complexes were resolved by SDS-PAGE and analyzed for Tctex-1 by Western blot analysis. As expected, antibody 70.1, directed against the intermediate chain of dynein, co-precipitated Tctex-1 (Fig. 8, lane 2), which is a light-chain subunit of dynein and is thought to interact directly with the intermediate chain (31, 37). An IgG control antibody, directed against mouse Nectin 2 (which is not expressed on human cells), did not precipitate any Tctex-1 protein (Fig. 8, lane 3). However, when incubated with anti-CD155 antibodies D171 and P44, Tctex-1 protein could be precipitated from the cell lysate (Fig. 8, lane 4). This observation indicated that CD155 and Tctex-1 also interacted specifically in intact cells.

DISCUSSION

In this study we have described the interaction between the cytoplasmic domain of the poliovirus receptor CD155 and Tctex-1, an association that we originally detected in a yeast-hybrid screen. We have provided evidence for the authenticity of this interaction by four independent biochemical and immunological assays in vitro and in intact cells. Our data indicate that Tctex-1 interacts with an SKCSR motif in the juxtamembrane region of the cytoplasmic domain of CD155. We have, furthermore, presented evidence of the presence of CD155 protein in neurons of the spinal cord, particularly motor neurons, and along fiber tracts within the dorsal root and the sciatic nerve. The specific interaction between CD155 and Tctex-1 can serve to explain an enduring conundrum: How does poliovirus receptor mediate retrograde transport of the entire vesicle via the dynein complex to the motor neuron cell body (Fig. 9). Indeed, our evidence of Tctex-1 localization to small punctae within motor neurons and peripheral nerves, reminiscent of membrane-bound organelles, would agree with such a model. This scenario requires the presence of Tctex-1 near the presynaptic membrane where, according to our model, virus uptake would occur. Interestingly, Chuang et al. (35) found Tctex-1 to be concentrated in small pre-synaptic vesicles at axon terminals of subsets of rat hippocampal neurons. Although it has not been directly shown at the subcellular level that Tctex-1 is expressed at motor axon terminals, Mou and colleagues (32) found the protein to be highly enriched in the eletroplax of Torpedo californica, an organ resembling a large specialized neuromuscular junction. Together with our evidence (Fig. 5) and that of others (34) of Tctex-1 immune reactivity within the sciatic nerve, it is plausible to speculate that Tctex-1 may serve a function in retrograde transport from the motor axon terminal to the neuronal cell body. The recent findings of Yano and colleagues (34) lend further support to this hypothesis. This group showed that retrograde axonal transport of neurotrophin receptors (TrkA, B, C) within the sciatic nerve of rats is facilitated by association of Trk with the dynein motor complex via binding of Tctex-1 (34). Thus, neu-
Nectin-1, Nectin-2, as well as CD155 for cell entry (49, 50). PRV is being used widely as a transneuronal tracer in neuroanatomical studies by virtue of monitoring its retrograde travel from the site of injection along axonal pathways of neurons (51, 52). The P protein of rabies and Mokola virus has been found to bind to the dynein light-chain polypeptide LC8 (46, 47). Indeed, CD155 as well as acetylcholine receptor, a putative receptor for rabies virus, are expressed at the NMJ (43, 53). Just as in provocation poliomylitis, rabies virus pathogenesis is mostly preceded by muscle trauma (here, the bite of a rabid animal) with subsequent virus entry into axons and retrograde transport to neuronal targets (54).

It appears that the underlying mechanisms of CNS invasion for all three groups of viruses, poliovirus, lyssaviruses (e.g., rabies), and herpesviruses (herpes simplex virus, PRV), are similar by hijacking the dynein machinery for retrograde travel to target neurons.

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CD155 Interaction with Dynein

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Steffen Mueller, Xuemei Cao, Reinhold Welker and Eckard Wimmer

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