JOURNAL OF VIROLOGY, Sept. 2004, p. 9564–9567 Vol. 78, No. 17
0022-538X/04/$08.00+0 DOI: 10.1128/JVI.78.17.9564–9567.2004

The Central Half of Pit2 Is Not Required for Its Function as a Retroviral Receptor

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Received 24 February 2004/Accepted 19 April 2004

The type III sodium-dependent phosphate (NaP_i) cotransporter, Pit2, is a receptor for amphotropic murine leukemia virus (A-MuLV) and 10A1 MuLV. In order to determine what is sufficient for Pit2 receptor function, a deletion mutant lacking about the middle half of the protein was made. The mutant supported entry for both viruses, unequivocally narrowing down the identification of the sequence that is sufficient to specify the receptor functions of Pit2 to its N-terminal 182 amino acids and C-terminal 170 amino acids.

The type III sodium-dependent phosphate (NaP_i) symporter, Pit2, from humans is a receptor for amphotropic murine leukemia virus (A-MuLV) and the related strain 10A1 MuLV (13, 22, 23, 25, 40, 44). The related Pit1 protein from humans (62% amino acid identity) is a receptor for gibbon ape leukemia virus, feline leukemia virus subgroup B, and 10A1 MuLV (22, 24, 39). The two human type III NaP_i symporters show about 25% amino acid identity with the *Neurospora crassa* NaP_i symporter Pho-4 (11, 40). Several studies have aimed at identifying sequences that are critical for the viral receptor functions of Pit1 and Pit2. These studies used chimeras between Pit1 and Pit2 orthologs, Pit1 orthologs, human Pit1 and Pho-4, human Pit2 and Pho-4, and human Pit1 and Pit2 and Pho-4 as well as Pit1 and Pit2 mutants (3, 5–8, 12, 14–16, 22, 26–28, 31–33, 38, 42, 43). The results suggest that amino acids and/or sequences present in human Pit2 positions 67 to 91 (8), 107 to 141 (14), 517 to 530 (15), and 522 to 530 (28) or corresponding positions in related proteins (15, 16, 22, 28, 33) specify or are involved in A-MuLV receptor function. Moreover, human and mouse Pit1 chimera studies suggest that amino acids in the mouse and human Pit1 regions corresponding to human Pit2 sequence 522 to 530 also are involved in 10A1 receptor function (16); these results have been confirmed in studies of human Pit2 mutants (unpublished results).

The design of the majority of the chimerical and mutant proteins (14–16, 22, 28, 33) was based on a membrane topology model for Pit1, Pit2, and Pho-4, which was suggested by Johann and coworkers on the basis of Kyte-Doolittle hydropathy plots (4, 11, 40) (Fig. 1B). Recently, new topology models that differ from the previous models have been proposed for Pit1 (7) and Pit2 (30) (Fig. 1A). If Pit1 and Pit2, however, exhibit different topologies in the membrane, the interpretation of which sequences are directly involved in receptor function for A-MuLV and 10A1 based on results obtained with Pit1/Pit2 chimeras may be incorrect. Moreover, one cannot in general exclude the possibility that amino acid exchanges in, for example, one part of Pit2 would lead to overall structural changes in another part of the protein, as shown for Pit1 by Farrell et al. (7). Results obtained with exchange mutants, therefore, may also potentially lead to incorrect interpretations concerning the identities of amino acids in Pit2 that are directly involved in A-MuLV and 10A1 receptor functions. It is obvious that these problems cannot be addressed simply by extending the use of chimeric Pit1/Pit2 proteins or exchange mutants of Pit2; however, functional deletion mutants can better identify which Pit2 sequences are directly involved in specifying receptor function.

We hypothesized that if the A-MuLV–and possibly 10A1–receptor determinants are indeed positioned in the N- and/or C-terminal ends of Pit2, it may be possible to delete the middle part of Pit2 and still retain its receptor functions. Accordingly, we made a mutant human Pit2 protein, $Pit2\Delta L_{183}\text{-}V_{483}$, in which the part between arginine 182 and histidine 484 was deleted in the 652-amino-acid protein.

The $Pit2\Delta L_{183}\text{-}V_{483}$ mutant was made from the pcDNA1A^{R}tkpA-derived expression plasmid pOJ74 (Wyeth-Ayerst Research, Pearl River, N.Y.) encoding human Pit2 (40) by using the forward primer ATGGCTGGGGAAGTTA GTGC and the reverse primer GGGTTACCGGAGGCCCG TGTGGAGGACAAGGTA; the latter was used to create the link between the 5′ sequence encoding $PNGLRA_{182}$ and the 3′ sequence encoding H_{484}LLFH (Fig. 1). The PCR amplification product was digested with Bsu36I and Sse8387I and used to replace the corresponding fragment in plasmid pOJ74, resulting in a plasmid encoding the mutant $Pit2\Delta L_{183}\text{-}V_{483}$ protein. The authenticity of the nucleotide sequence was confirmed. The ability of $Pit2\Delta L_{183}\text{-}V_{483}$ to support A-MuLV and 10A1 entry was compared to that of human Pit2 by using a transient transfection-infection assay and A-MuLV and 10A1 vector pseudotypes derived from the packaging cell lines PA317 (19, 21) and PT67 (20), respectively, both carrying the G1BgSvN transfer vector (18) as previously described (2, 28). The G1BgSvN transfer vector is a Moloney MuLV-based vector that expresses LacZ and neomycin phosphotransferase (18). The titers of A-MuLV and 10A1 vector stocks were determined on dog D17 cells (ATCC CCL 183) as previously described (28) and were 1.4×10^5 to 4.7×10^5 and 0.7×10^5 to 6.0×10^5 CFU per ml, respectively; however, the stocks were diluted to 40,000 infectious vectors per 1.5 ml before use. Briefly, hamster CHO K1 cells (ATCC CCL 61), before the

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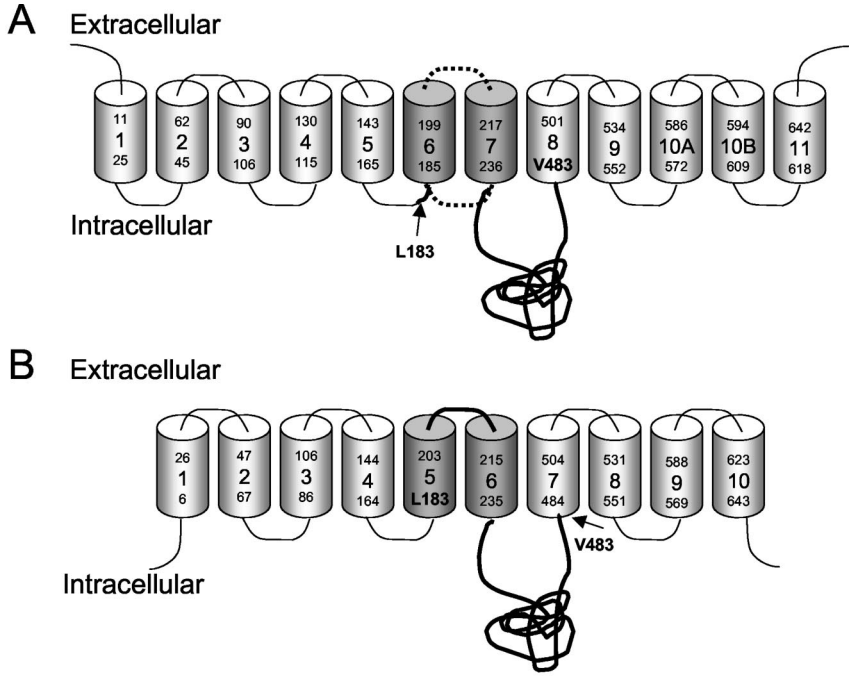

FIG. 1. Putative membrane topology models of Pit2 based on Salaün et al. (30) (A) and Johann et al. (11, 40) (B); see references for details on the models. The membrane topology model of Pit2 after Salaün et al. (30) was based on (i) epitope tagging of N- and C-terminal ends, (ii) orientation of microsomal membrane-inserted in vitro transcribed and translated C-terminal-truncation mutants, and (iii) glycosylation of wild-type human Pit2 and lack of glycosylation of a human $Pit2N_{81}V$ mutant in whole-cell extracts from CHO K1 cells overexpressing Pit2 and $Pit2N_{81}V$. Salaün and coworkers did not present functional studies (e.g., viral receptor function) of the C-terminal truncation mutants (30). For comparison, the first proposed membrane topology model of Pit2 based on Kyte-Doolittle hydropathy plots is shown (11, 40) in panel B. The middle part of human Pit2 including amino acid 183 to amino acid 483 (the sequences highlighted in both A and B) is deleted in the mutant $Pit2\Delta L_{183}\text{-}V_{483}$.

fifth passage and kept subconfluent during cultivation, were seeded in dishes with a diameter of 60 mm. These cells were transfected with 1 μg of plasmid DNA encoding human Pit2 or equimolar amounts of plasmid encoding $Pit2\Delta L_{183}\text{-}V_{483}$. Mock-treated cells were transfected with empty vector DNA. The total amounts of DNA in the transfection solutions were kept constant by using plasmid pUC19 as carrier DNA. At 48 h posttransfection, 1.5 ml of diluted vector stocks was added per dish in the presence of Polybrene. After 48 h, the dishes were fixed and stained, and the number of β-galactosidase-positive (infected) cells per dish was either counted (Table 1) or visualized (Fig. 2). Note that in each experiment shown in Table 1, three independent transfection solutions were made per construct, and each solution was evaluated for its ability to support both A-MuLV and 10A1 infection (experiments 1 and 2) or 10A1 infection (experiment 3); thus, the numbers shown in Table 1 represent the average levels of infection from three 60-mm dishes that received independent transfection solutions.

Even though approximately the middle half of the Pit2 protein is deleted in $Pit2\Delta L_{183}\text{-}V_{483}$, the mutant sustained receptor function for both A-MuLV and 10A1 in the ranges of 13 to 25% and 10 to 24%, respectively, of the infection levels supported by wild-type human Pit2 (Table 1). No infection by 10A1 or A-MuLV vector pseudotypes was observed in mock-transfected CHO K1 cells (Table 1 and Fig. 2). We occasionally observe a low background level of infection with these vector pseudotypes on our CHO K1 cells (<2 to 6 blue cells per 60-mm dish or <2 CFU/ml) (reference 15 and our unpublished observations) although such an occurrence is the exception (Table 1 and Fig. 2) (2, 15, 28). It should be noted, however, that there exist CHO K1 subpopulations or subclones that are susceptible to 10A1 infection (9, 22).

While the results presented here do not provide new infor-

TABLE 1. Permissivity for infection of CHO K1 cells transiently transfected with vector expressing Pit2 or $Pit2\Delta L_{183}\text{-}V_{483}$ or empty vector[a]

Construct[b]	No. (%) of cells infected[c]				
	A-MuLV		10A1 MuLV		
	Expt 1	Expt 2	Expt 1	Expt 2	Expt 3
Pit2 (pOJ74)	100 ± 22	100 ± 8	100 ± 5	100 ± 8	100 ± 9
$Pit2\Delta L_{183}\text{-}V_{483}$	25 ± 1	13 ± 2	10 ± 1	12 ± 4	24 ± 3
Empty vector[d]	<0.0008	<0.002	<0.002	<0.001	<0.001

[a] The experimental setup is described in the text. A-MuLV and 10A1 vector pseudotypes were tested on the same DNA precipitates of a given construct in experiment 1 (one independent set of DNA preparations) and in experiment 2 (another independent set of DNA preparations). In experiment 3, only the 10A1 vector pseudotype was tested (same DNA preparations as in experiment 1).

[b] Receptor and mutant receptor sequences were cloned into $pcDNA1A^{R}tkpA$.

[c] The data are averages of three independent transfections ± standard errors of the means. The average number of blue cells per three 60-mm dishes transfected with a plasmid expressing Pit2 was assigned a value of 100% (42,000, 19,000, 18,000, 32,000, and 32,000 blue cells per dish for A-MuLV in experiments 1 and 2 and for 10A1 in experiments 1, 2, and 3, respectively).

[d] Values are based on the detection limit of 1 blue cell per three 60-mm-diameter dishes.

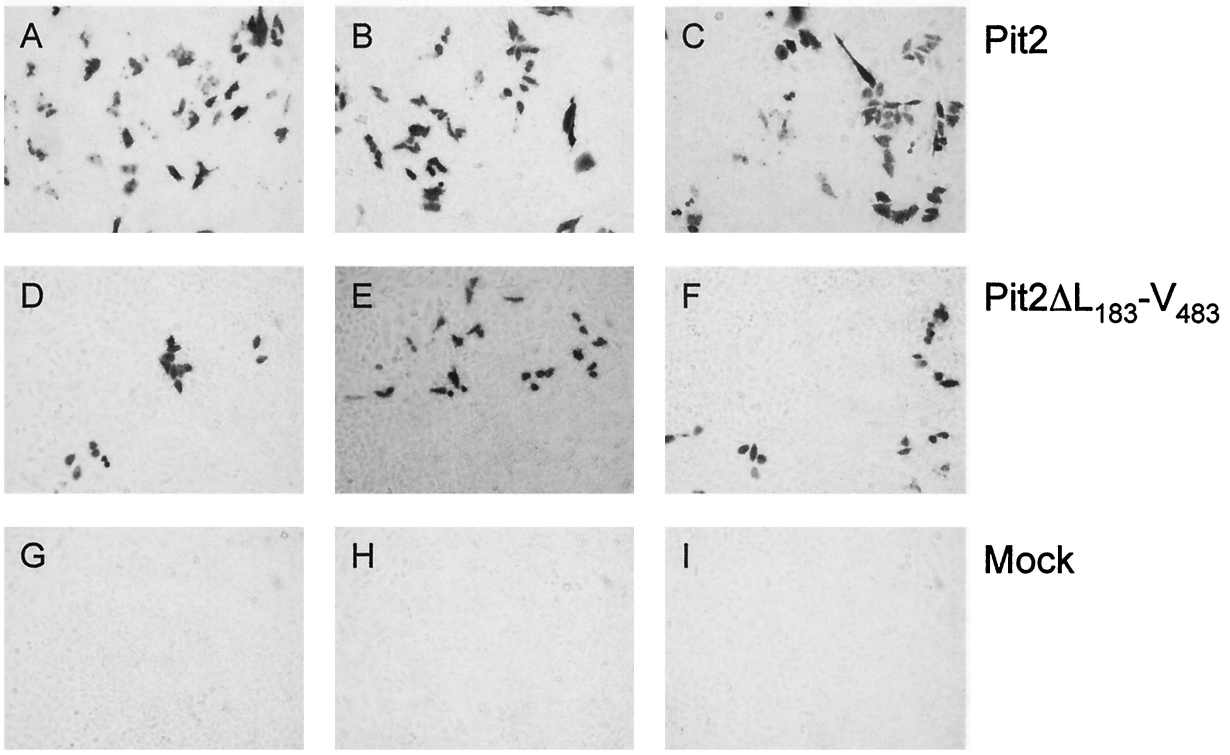

FIG. 2. Susceptibilities of transfected CHO K1 cells. Transfections were performed as described in the text. Panels A to C show three dishes receiving independent DNA precipitates of the plasmid encoding Pit2, panels D to F show three dishes receiving independent DNA precipitates of the plasmid encoding Pit2ΔL_{183}-V_{483}, and panels G to I show three dishes receiving independent DNA precipitates of empty expression vector (Mock). The experiment was performed according to the method shown in Table 1; however, a third independent set of DNA preparations was used. At 48 h posttransfection, cells were challenged with PT67-derived vector pseudotypes (10A1 MuLV pseudotypes) carrying the LacZ-expressing G1BgSvN transfer vector. Forty-eight hours after vector exposure, cells were fixed and stained for the presence of β-galactosidase-positive (infected) cells; infected cells are blue (dark). No blue cells were present in the mock-transfected cultures. Random fields from each plate in the triplicate setups are shown at a magnification of ×200.

mation on the membrane topology of Pit2 per se, the observation that Pit2ΔL_{183}-V_{483} supports A-MuLV and 10A1 infection shows that the N-terminal 182 amino acids and C-terminal 170 amino acids of human Pit2 are sufficient for specifying A-MuLV and 10A1 receptor functions. Thus, the results of the present study narrow the viral binding domains and other possible sequences directly involved in A-MuLV and/or 10A1 entry to sequences positioned in these N- and C-terminal regions of human Pit2. The regions include all Pit2-specific sequences previously identified as influencing Pit2 receptor function for A-MuLV and 10A1 (8, 14–16, 22, 28, 33), and the Pit2ΔL_{183}-V_{483} mutant may prove to be a powerful tool in identifying whether all of these sequences, indeed, are directly involved in A-MuLV binding and entry. The mutant lacks a sequence originally identified as a topogenic determinant in Pit1 (region B) and shown to affect the results of Pit1/Pit2 chimerical studies (7); however, a number of related putative phosphate symporters from archaea and bacteria (e.g., U15187, AL939110, AP001512, AE000978, AE013582 [National Center for Biotechnology Information accession numbers]) resemble Pit2ΔL_{183}-V_{483}, which suggests that the mutant constitutes a structural unit here shown to specify viral receptor function. It is therefore also possible that the mutant will sustain NaP_i transport. It should be noted, however, that there is no correlation between the ability of Pit2 to transport phosphate and support viral entry (2), and the uncoupling of transport and receptor functions has also been shown for two other transporters with retroviral receptor function (37, 41) as thoroughly discussed in a recent review (36). Indeed, like Pit2, a number of the identified receptors for retroviruses are polytopic solute transporters (1, 10, 17, 24, 29, 34, 35). The present study shows that it is possible to use a deletion mutant of Pit2 for studying its viral receptor function and raises the possibility that deletion mutants of other polytopic proteins may also provide insight into which receptor sequences are directly involved in receptor functions for their cognate viruses.

We thank Bryan O'Hara for plasmid pOJ74 and Maribeth V. Eiden for the PA317GBN cell line.

This study was supported by the Lundbeck Foundation, the Novo Nordisk Foundation, the Danish Medical Research Council (grant 9802349), the Karen Elise Jensen Foundation, and the Danish Cancer Society (grant DP00092).

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