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# Use of Vesicular Stomatitis Virus Pseudotypes to Map Viral Receptor Genes: Assignment of RD114 Virus Receptor Gene to Human Chromosome 19

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Vesicular stomatitis virus pseudotypes bearing envelope glycoproteins of the endogenous feline type C retrovirus, RD114, were used to assay the expression of receptors specific to RD114 on the surfaces of mouse-human hybrid cells carrying different human chromosomes. These studies show that the gene encoding the RD114 receptor is located on human chromosome 19.

Viral infection, either lytic or latent, of any cell type is dependent on an initial interaction occurring at the cell surface which results in viral penetration of the cellular membrane. In the case of both enveloped and nonenveloped viruses, examination of this interaction has demonstrated recognition between the structural proteins expressed on the surface of viral particles and cell surface components (viral receptors) (1). Expression of these membrane receptors is dependent on the presence and functional expression of cellular structural genes.

In previous studies, assignment of these genes to specific chromosomes has been established by examination of the interaction of virus, viral proteins, or both with hybrid cell lines (10, 13, 15). Each hybrid line consists of a genome of the entire chromosomal composition of a resistant cell type and a specific, but incomplete set of chromosomes of the sensitive cell type. The chromosomes present are identified cytologically (karyotype analysis) and by demonstration of the expression of specific isozymes whose gene locations are already known. Determination of the presence of the viral receptor has been dependent on the demonstration of the susceptibility of individual hybrid cell lines to viral infection (4, 10, 13, 15) or the binding of viral products to cellular surfaces (1). These methods, however, suffer from several significant limitations: (i) viral infection, although requiring the functional presence of viral receptors, is also dependent upon subsequent viral gene expression; (ii) the virus-cell interaction determined by binding assays may not be specific, nor biologically significant; (iii) measurement of the binding of viral products provides no assessment of the functional status of the viral receptors.

To overcome these limitations, pseudotype viruses have been employed for the study of cell surface viral receptors. Pseudotype virus particles are viral "hybrids" which commonly arise during dual infection of susceptible cells by a variety of enveloped viruses. They are composed of the genome of one virus and the coat of a second virus. Adsorption to and penetration of cells by these pseudotype particles is dependent on the presence of cell receptors for the virus providing the envelope antigens. Demonstration of this event, however, is independent of any subsequent functional activity of the virus donating the envelope component but instead requires the expression of the enclosed genome of the second virus. By employing an appropriate lytic virus as genome donor, expression of this virus will result in cell death with release of more lytic virus, leading to infection of surrounding cells and eventual plaque production. Hence the presence of cell receptors for the envelope antigens of the first virus may be monitored by observing the development of lytic plaques resulting from genome expression of the second virus. Furthermore, these plaques should be observed only in those cell types possessing the surface receptor for the first virus (envelope donor).

Vesicular stomatitis virus (VSV), a member of the rhabdovirus group of enveloped viruses, has been used extensively as the genome donor in pseudotype studies because it is capable of replicating in a wide variety of cell types with the rapid production of plaques (20). Additionally, its ability to produce pseudotypes with representative members of all groups of enveloped viruses has permitted its use in a wide range of investigations, including previous studies of the expression and genetic control of receptors for

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avian type C retroviruses in avian and mammalian cells (2) and the examination of receptors for murine ecotropic retroviruses in a variety of mouse-hamster hybrid cell lines (8, 17). This latter study, similar in design to that described in this report, has permitted assignment of the gene responsible for expression of the cellular receptor for this virus to mouse chromosome 5.

Several type C retroviruses (RD114, murine leukemia virus type X, simian sarcoma-associated virus, feline leukemia virus type C) are known to replicate efficiently in human cells but to be unable to infect murine cells (11, 12, 14, 19). A previous study of VSV pseudotypes of these viruses showed that the inhibition of infection observed with murine cells occurs at the level of viral penetration, presumably due to lack of expression of the appropriate viral receptors (18). Since human cells must express such receptors, examination of different mouse-human hybrid cell lines permits correlation between the presence of the viral receptor and the human chromosome(s) which codes for the receptor structural gene.

Such studies utilizing pseudotypes of VSV containing the envelope antigens of RD114, an endogenous type C retrovirus of cats, have been undertaken, and the results are presented in Tables 1 and 2, along with the karyotype analysis of each hybrid cell line examined. Isozyme analysis was also conducted on these cell lines (Table 3). Six hybrid cell lines demonstrated plaques when infected with this pseudotype, and nine were resistant. The only human chromosome common to the susceptible lines was chromosome 19. This chromosome was found to be absent in the resistant cell lines. Among the plaque-positive clones, chromosome 19 was not demonstrable karyotypically in clone AHA-3d-14, although this clone was clearly positive for the human isozyme glucose phosphate isomerase, which is determined by a gene located on chromosome 19 (4). Conversely, the glucose phosphate isomerase analysis of clone IL-115 was uncertain, although the karyotypic presence of chromosome 19 was unequivocal. The two discordant clones for this assignment are explained by the fact that a chromosome was considered present for the analysis if it was observed in at least 10% of the karyotyped cells, whether or not it was detected by isozyme analysis. In the + − discordancy, chromosome 19 was not observed in 20 cells, although its isozyme (glucose phosphate isomerase) was detected. Chromosome 19 was observed in 4 of 20 cells in the − + discordancy, but its marker isozyme was absent. The chromosome identified in this cell line as human 19 may actually have been a mouse chromosome which resembled it. Taking these data together, the correlation of the presence or absence of specific human chromosomes with cellular susceptibility to viral infection as presented in Tables 2 and 3 confirms the strong and specific association of this property with chromosome 19.

These results assign the human gene responsible for the expression of the cell receptor for RD114 virus to chromosome 19. Because these studies are based on the demonstration of susceptibility of hybrid cells to infection by VSV pseudotypes of RD114 rather than RD114 itself, this assay is independent of RD114 gene expression after viral penetration and is a measure solely of the interaction of RD114 envelope antigens with cell surface structures. The biological and functional integrity of this interaction is demonstrated by the release of the enclosed VSV genome in functional form, evidenced by plaque formation.

The fact that functional expression of the viral receptor could be detected in hybrid cell lines containing human chromosome 19 demonstrates that expression of this human gene is dominant in these hybrids. Furthermore, the fact that cell lines possessing chromosome 19 are susceptible to pseudotype infection suggests that, under conditions of this assay, expression of this gene is either constitutive or else subject to regulation by another gene(s) also present on chromosome 19. Whether expression of the receptor function is regulated under natural conditions by independently segregating genes is not known, but examples of this type of regulation, as well as constitutive gene expression, have been described for avian type C retrovirus receptors in avian cells (16).

As VSV will produce pseudotypes with an extremely wide range of enveloped viruses it should be possible, by use of the techniques described above, to assign chromosomal location to genes coding for receptors for a variety of different viruses. In the case of closely related viruses, it would be of interest to determine whether these genes are located on different chromosomes and whether they are independently controlled and expressed. Preliminary observations with feline leukemia virus type C, an exogenous type C retrovirus of cats, suggest independent segregation of genes responsible for expression of cell receptors for feline type C retroviruses, because no positive correlation has been seen between human chromosome 19 and susceptibility to infection. Studies with avian retroviruses using classical Mendelian methods have also demonstrated independent segregation of receptor genes for subgroup A and B viruses (7).

Chromosome 19 contains several other genes

TABLE 1. *Chromosome frequency and susceptibility to RD114 pseudotypes in human-mouse hybrids*[a]

| Cell line | Plaque production | Chromosome | | | | | | | | | | | | | | | | | | | | | | | No. of cells karyotyped |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | X | Y | |
| *ts*Cl AGOH | − | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| A9 | − | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| GM 589 | + | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 0 |
| AHA-3d-14 | + | 0 | 0 | 90 | 75 | 0 | 0 | 0 | 0 | 0 | 70 | 85 | 0 | 0 | 0 | 0 | 0 | 0 | 80 | 0 | 0 | 0 | 0 | 0 | 0 | 20 |
| IL-115 | + | 0 | 0 | 0 | 0 | 0 | 0 | 68 | 0 | 0 | 0 | 0 | 65 | 3 | 0 | 23 | 0 | 71 | 0 | 42 | 0 | 13 | 0 | 3 | 0 | 31 |
| IL-115-4d | + | 0 | 0 | 0 | 0 | 0 | 0 | 20 | 0 | 0 | 0 | 0 | 21 | 0 | 0 | 0 | 0 | 60 | 0 | 10 | 0 | 0 | 0 | 0 | 0 | 70 |
| BDA-17b#17 | + | 74 | 58 | 71 | 61 | 58 | 56 | 0 | 0 | 0 | 0 | 0 | 0 | 61 | 3 | 42 | 61 | 71 | 61 | 63 | 66 | 84 | 53 | 79 | 0 | 38 |
| BDA-17b#6 | + | 0 | 7 | 24 | 32 | 7 | 0 | 0 | 28 | 0 | 4 | 24 | 0 | 0 | 11 | 4 | 11 | 75 | 14 | 28 | 18 | 64 | 7 | 50 | 0 | 28 |
| 11-41pT-8 | + | 0 | 0 | 0 | 27 | 27 | 0 | 18 | 0 | 0 | 0 | 91 | 44 | 18 | 44 | 9 | 18 | 44 | 27 | 54 | 18 | 64 | 73 | 82 | 0 | 11 |
| AIM-15a | − | 65 | 50 | 0 | 0 | 55 | 0 | 75 | 10 | 0 | 0 | 75 | 45 | 0 | 25 | 40 | 15 | 70 | 55 | 20 | 35 | 70 | 0 | 20 | 0 | 20 |
| 163ApT-1 | − | 36 | 0 | 79 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 95 | 32 | 0 | 21 | 21 | 0 | 0 | 53 | 0 | 0 | 0 | 53 | 0 | 0 | 19 |
| WAIVa-DAP | − | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 48 | 0 | 0 | 50 |
| BDA-10a-3 | − | 0 | 40 | 57 | 60 | 0 | 40 | 0 | 40 | 0 | 67 | 73 | 93 | 40 | 0 | 0 | 0 | 50 | 0 | 7 | 60 | 0 | 0 | 37 | 10 | 30 |
| BDA-10a-4a | − | 0 | 10 | 60 | 40 | 0 | 65 | 0 | 5 | 60 | 60 | 75 | 80 | 50 | 30 | 0 | 25 | 45 | 0 | 5 | 0 | 0 | 0 | 70 | 0 | 20 |
| AIM-15a-B1 | − | 0 | 55 | 0 | 0 | 40 | 0 | 50 | 30 | 0 | 0 | 90 | 10 | 10 | 10 | 10 | 0 | 75 | 55 | 0 | 0 | 60 | 0 | 0 | 0 | 20 |
| AHA-11a | − | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 99 | 0 | 100 |
| WAV-R4d-F9-4a | − | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 98 | 0 | 0 | 0 | 100 |
| 157BnpT-4 | − | 0 | 0 | 47 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 81 | 0 | 0 | 58 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 75 | 0 | 60 |

[a] Plaque production by RD114 pseudotype of VSV in individual cell lines was determined as previously described (14). The inoculum utilized had a titer of $10^4$ PFU/ml when assayed on the parental human cells. Cell lines demonstrating 30 plaques were considered positive (+) for plaque production, whereas those reported as negative (−) consistently failed to demonstrate more than three plaques when tested under identical conditions. From experiments with differing ratios of susceptible to resistant cells, fewer than 10% susceptible cells could not be regularly detected with the assay. The number refers to the percentage of cells which contained the human chromosome. All cell lines were maintained in Dulbecco-Vogt medium supplemented with 5% fetal bovine serum. Clones *ts*Cl AGOH, 11-41pT-8, and 163ApT-1, which contain a temperature-sensitive mutation, were grown at 34°C. The rest of the lines were grown at 37°C. Two of the cell lines, *ts*Cl AGOH and A9, were mouse parental lines for the hybrid clones. GM 589 is a human fibroblast line used as the human parental cell line for many of the hybrid lines. All of the hybrid lines were derived from whole cell fusions utilizing inactivated Sendai virus. Karyotype analysis was performed on Giemsa-banded chromosomes. Hoechst staining and G-11 staining were also employed in chromosome identification.

whose functions are known. Glucose phosphate isomerase, peptidase D, and alpha-mannosidase-2 (5) have been mapped to this chromosome. In addition, the expression of the receptor for poliovirus has also been mapped to chromosome 19 (15). The poliovirus receptor, however, appears to be quite distinct from the receptor for RD114 virus. We have infected human cells with RD114 virus so that the receptors for this virus become functionally blocked as tested by subsequent challenge with the VSV(RD114) pseudotype. Such cells were still fully susceptible to lytic poliovirus infection (unpublished data).

The use of pseudotypes also permits a distinction to be made between those cellular genes responsible for viral receptor expression and those required for subsequent viral gene function. Previous investigations with baboon endogenous virus, a type C retrovirus, have indicated that human chromosomes 6 and 19 are required for viral infection and replication in human-hamster hybrid cells (3, 4, 9, 10). Since baboon endogenous virus utilizes the same receptors as RD114 in human cells to initiate infection (18), we may conclude that the chromosome 19 marker is the cell surface receptor identified here. However, the replication and integration of baboon endogenous virus have been shown to depend on the presence not of chromosome 19 but of chromosome 6 (9, 10). Whether RD114, a

TABLE 2. *Statistical analysis of the correlation between individual human chromosomes and susceptibility to RD114 pseudotypes in human-mouse hybrid clones*[a]

| Chromosome | No. of clones | | | | CC | Chi-square | Overall OR |
|---|---|---|---|---|---|---|---|
| | Plaque production + | | Plaque production − | | | | |
| | Chromosome + | Chromosome − | Chromosome + | Chromosome − | | | |
| 1 | 1 | 5 | 2 | 7 | −0.07 | 0.07 | 3.33 |
| 2 | 1 | 5 | 4 | 5 | −0.29 | 1.25 | 2.04 |
| 3 | 3 | 3 | 4 | 5 | 0.05 | 0.04 | 4.61 |
| 4 | 4 | 2 | 2 | 7 | 0.44 | 2.96 | 5.81 |
| 5 | 2 | 4 | 2 | 7 | 0.12 | 0.23 | 3.64 |
| 6 | 1 | 5 | 2 | 7 | −0.07 | 0.07 | 3.48 |
| 7 | 3 | 3 | 2 | 7 | 0.29 | 1.25 | 5.67 |
| 8 | 1 | 5 | 3 | 6 | 0.18 | 0.51 | 2.78 |
| 9 | 0 | 6 | 1 | 8 | −0.22 | 0.71 | 3.61 |
| 10 | 1 | 5 | 2 | 7 | −0.07 | 0.07 | 4.39 |
| 11 | 3 | 3 | 6 | 3 | −0.17 | 0.42 | 3.20 |
| 12 | 3 | 3 | 5 | 4 | −0.05 | 0.04 | 4.09 |
| 13 | 2 | 4 | 3 | 6 | 0.00 | 0.00 | 3.31 |
| 14 | 2 | 4 | 5 | 4 | −0.22 | 0.71 | 2.43 |
| 15 | 2 | 4 | 3 | 6 | 0.00 | 0.00 | 3.89 |
| 16 | 3 | 3 | 2 | 7 | 0.29 | 1.25 | 4.20 |
| 17 | 5 | 1 | 4 | 5 | 0.39 | 2.27 | 5.48 |
| 18 | 4 | 2 | 3 | 6 | 0.33 | 1.61 | 5.11 |
| 19 | 5 | 1 | 1 | 8 | 0.72 | 7.82 | 7.25 |
| 20 | 3 | 3 | 2 | 7 | 0.29 | 1.25 | 4.25 |
| 21 | 4 | 2 | 3 | 6 | 0.33 | 1.61 | 5.02 |
| 22 | 3 | 3 | 2 | 7 | 0.29 | 1.25 | 5.71 |
| X | 3 | 3 | 5 | 3 | −0.12 | 0.22 | 2.41 |
| Y | 0 | 6 | 0 | 9 | 0.00 | 0.00 | 4.22 |

[a] Statistical analysis was performed on the results with the aid of a computer program, ASSIGN, developed to analyze data derived from somatic cell hybrids for gene mapping purposes (6). Plaque production was compared with chromosome constitution in each of the hybrid clones. Analysis involves the assignment of a gene to a chromosome. Three statistical procedures were employed for chromosome assignment. CC, Coefficient of correlation. The possible range for coefficient of correlation is −1 to +1. A perfect association between plaque production and a specific chromosome yields a value of +1. Zero indicates no association, and −1 indicates perfect negative association. Chi-square as employed here is not a test of significance; rather, it is a measure of dependence and is calculated for each chromosome independently of all other chromosomes. The overall OR statistic considers all of the chromosome data simultaneously. Each clone is assigned a value of 100. In clones that yield plaques, the 100 points are divided equally among all chromosomes present in the clone. In clones that do not yield plaques, the 100 points are divided equally among those chromosomes not detected in the clone. The scores are added for each chromosome and then divided by the number of hybrid clones assayed for plaque production. The chromosome with the highest score is considered to be the most likely candidate coding for the viral receptor. Human chromosome 19 had the highest value in each of the three statistical analyses.

TABLE 3. *Isozyme analysis and susceptibility to RD114 pseudotypes in human-mouse hybrids*[a]

| Chromosome | Isozyme[b] | No. of clones: Plaque production + | | Plaque production − | | Concordancy ++, −− | Discordancy +, +− | Chi-square |
|---|---|---|---|---|---|---|---|---|
| | | Isozyme + | Isozyme − | Isozyme + | Isozyme − | | | |
| 1 | PGM-1 | 1 | 4 | 3 | 6 | 7 | 7 | 0.008 |
| | PEP C | 1 | 2 | 1 | 6 | 7 | 3 | 0.030 |
| 2 | IDH | 1 | 5 | 4 | 4 | 5 | 9 | 0.063 |
| | MOR | 1 | 4 | 2 | 5 | 6 | 6 | 0.114 |
| 3 | — | — | — | — | — | — | — | — |
| 4 | — | — | — | — | — | — | — | — |
| 5 | HEX B | 1 | 5 | 0 | 8 | 9 | 5 | 0.022 |
| 6 | MOD | 1 | 5 | 2 | 6 | 7 | 7 | 0.080 |
| 7 | UP | 1 | 0 | 2 | 4 | 5 | 2 | 1.556 |
| | β-GLUC | 0 | 5 | 0 | 3 | 3 | 5 | NA[c] |
| 8 | GR | 0 | 5 | 0 | 3 | 3 | 5 | NA |
| 9 | AK-1 | 1 | 4 | 0 | 6 | 7 | 4 | 0.009 |
| 10 | GOT-1 | 1 | 4 | 2 | 6 | 7 | 6 | 0.219 |
| | ADOK | 1 | 2 | 0 | 6 | 6 | 2 | 0.140 |
| 11 | LDH A | 3 | 2 | 5 | 2 | 5 | 7 | 0.043 |
| 12 | TPI | 4 | 2 | 5 | 2 | 6 | 7 | 0.174 |
| | PEP B | 4 | 2 | 5 | 4 | 8 | 7 | 0.012 |
| 13 | ES-D | 0 | 5 | 1 | 5 | 5 | 6 | 0.009 |
| 14 | NP | 2 | 3 | 5 | 4 | 6 | 8 | 0.000 |
| 15 | MPI | 3 | 3 | 2 | 6 | 9 | 5 | 0.000 |
| 16 | APRT | 2 | 3 | 3 | 4 | 6 | 6 | 0.245 |
| 17 | GAL K | 1 | 3 | 3 | 2 | 3 | 6 | 0.141 |
| 18 | PEP A | 1 | 2 | 2 | 6 | 7 | 4 | 0.234 |
| 19 | GPI | 5 | 1 | 0 | 9 | 14 | 1 | 7.813 |
| 20 | ADA | 2 | 3 | 1 | 6 | 8 | 4 | 0.114 |
| 21 | SOD-1 | 5 | 1 | 3 | 5 | 10 | 4 | 1.367 |
| 22 | — | — | — | — | — | — | — | — |
| X | HPRT | 2 | 2 | 2 | 3 | 5 | 4 | 0.141 |
| | G6PD | 2 | 3 | 5 | 2 | 4 | 8 | 0.245 |
| | PGK | 2 | 2 | 5 | 2 | 4 | 7 | 0.004 |
| Y | — | — | — | — | — | — | — | — |

[a] Plaque production was compared with isozyme constitution for a test of synteny. Glucose phosphate isomerase, which is located on human chromosome 19, has the highest concordance with plaque production by RD114 pseudotypes. The one discordant clone was IL-115, in which 42% of the cells had chromosome 19, although the isozyme analysis was uncertain.

[b] Abbreviations: PGM-1, phosphoglucomutase-1; PEP C, PEP B, PEP A, peptidase C, B, and A, respectively; IDH, isocitrate dehydrogenase; MOR, malate oxidoreductase; HEX B, HEX A, hexosaminidase B and A, respectively; MOD, malate oxidoreductase-decarboxylating; UP, uridine phosphorylase; β-GLUC, β-glucuronidase; GR, glutathione reductase; AK-1, adenylate kinase-1; GOT-1, glutamate oxidoreductase-1; ADOK, adenosine kinase; LDH A, lactate dehydrogenase A; TPI, triose phosphate isomerase; ES-D, esterase-D; NP, nucleoside phosphorylase; MPI, mannose phosphate isomerase; APRT, adenine phosphoribosyltransferase; GAL K, galactokinase; GPI, glucose phosphate isomerase; ADA, adenosine deaminase; SOD-1, superoxide dismutase-1; HPRT, hypoxanthine phosphoribosyltransferase; G6PD, glucose 6-phosphate dehydrogenase; PGK, phosphoglycerol kinase.

[c] NA, Test not applicable.

virus which shares some genome homology with baboon endogenous virus, also requires the presence of chromosome 6 for replication is not known.

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