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Nucleotide Sequences of Feline Sarcoma Virus Long Terminal Repeats and 5′ Leaders Show Extensive Homology to Those of Other Mammalian Retroviruses

ANNIE HAMPE,[1] MARCELLE GOBET,[1] JOS EVEN,[2] CHARLES J. SHERR,[2]* AND FRANCIS GALIBERT[1]

Laboratoire d'Hematologie Experimentale, Centre Hayem, Hôpital Saint Louis, 75475 Paris Cedex 10, France,[1] and Viral Pathology Section, Laboratory of Tumor Virus Genetics, National Cancer Institute, Bethesda, Maryland 20205[2]

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The nucleotide sequences of the Gardner-Arnstein feline sarcoma virus (FeSV) long terminal repeat and the adjacent leader sequences 5′ to the viral *gag* gene were determined. These were compared with homologous portions of Snyder-Theilen FeSV and with previously published sequences for Moloney murine sarcoma virus and simian sarcoma virus proviral DNA. More than 75% of the residues in the FeSV R and U5 regions were homologous to sequences within the same regions of the other viral long terminal repeats. Unexpectedly, alignment of the FeSV sequences with those of the Moloney murine sarcoma and simian sarcoma viruses showed similar extents of homology within U3. The homologous U3 regions included the inverted repeats, a single set of putative enhancer sequences, corresponding to a "72-base-pair" repeat, and sequences, including the CAT and TATA boxes, characteristic of eucaryotic promotors. The 5′ leader sequences of both FeSV strains included a binding site for prolyl tRNA and a putative splice donor sequence. In addition, the FeSV leader contained a long open reading frame which was adjacent to and in phase with the ATG codon at the 5′ end of the FeSV *gag* gene. The open reading frame could code for a signal peptide of about 7.4 kilodaltons. Our results support the concept that the virogenic portions of both FeSV and simian sarcoma virus were ancestrally derived from viruses of rodent origin, with conservation of regulatory sequences as well as the viral structural genes.

A characteristic difference between the proviral DNA and genomic RNA of retroviruses is the presence of long terminal repeats (LTRs) at each proviral DNA terminus (20, 37) which are formed during the process of reverse transcription (16). Each proviral LTR contains the sequences U3-R-U5, where U3 and U5 designate unique sequences derived from the 3′ and 5′ ends, respectively, of viral RNA, and R designates a short, terminally redundant sequence present at both termini of viral RNA. The LTRs are assumed to play a critical role in the integration of proviral DNA into the host chromosome and are known to contain regulatory sequences necessary for the transcription of viral RNA (9, 10, 21, 28, 31, 41, 42, 45–47).

Directly downstream of the 5′ LTR is a leader sequence which separates the LTR from viral genes encoding structural proteins. The leader begins with a sequence complementary to portions of a tRNA molecule used as the primer in proviral DNA synthesis (16). For all replicating viruses, and for some classes of replication-defective genomes, subgenomic mRNAs encoding information from the 3′ end of proviral DNA (18, 49) contain leader sequences spliced from the extreme 5′ end of the viral genome (32). A packaging signal for encapsidation of viral RNA into virions may also be located within the leader sequences (38).

We have now determined the primary nucleotide sequences of the LTR and 5′ leader of the Gardner-Arnstein (GA) strain of feline sarcoma virus (FeSV) (15) and compared these with the homologous region of the Snyder-Theilen (ST) FeSV strain (44). For these analyses, we used a previously obtained λ phage clone containing transforming GA-FeSV proviral DNA (14). In DNA transfection assays, the molecularly cloned proviral DNA transforms NIH/3T3 cells at efficiencies of up to 10^4 focus-forming units per μg of DNA. The 5′ ends of the cloned GA-FeSV molecules, including the regions subjected to sequencing analysis, were successfully substi-

tuted into replicating feline leukemia viruses (J. Even, D. Lowy, S. Anderson, A. Hampe, F. Galibert, and C. J. Sherr, unpublished data), and were previously used to activate human cellular proto-oncogene sequences (7). Together, these data show that the GA-FeSV LTR is active in promoting transcription and that the leader sequences can function in tRNA binding, splicing, and packaging of viral RNA.

Additional sequencing analyses were performed with biologically inactive ST-FeSV DNA molecules, cloned as unintegrated DNA intermediates (39). This clone contains a small deletion within the viral oncogene which alters the reading frame encoding the transforming polyprotein (16a). In addition, comparative sequencing analyses (see below) are consistent with the possibility that the ST-FeSV LTR contains two *Eco*RI restriction sites within the U3 region. Because *Eco*RI sites were used to molecularly clone linear ST-FeSV DNA molecules, the LTR sequences are probably incomplete and may themselves be inactive.

Nucleotide sequencing of both DNA clones was performed by the method of Maxam and Gilbert (26). Usually, about 10 pmol of DNA was restricted with a given endonuclease, and the fragments were dephosphorylated and labeled with [γ-^{32}P]ATP and polynucleotide kinase as previously described (19). Under certain circumstances, fragments with recessed 3′ ends were labeled with a α-^{32}P-labeled nucleotide triphosphate with DNA polymerase I (17). All restriction sites used as starting points were analyzed as internal sites within overlapping fragments. The sequences of both strands of both the 5′ and 3′ LTRs were determined, but for convenience only one LTR sequence is shown.

The sequence of the GA-FeSV LTR (482 residues) and the leader sequence preceding the viral *gag* gene (432 residues) are shown in Fig. 1. The numbers on the top line refer only to the positions of residues within GA-FeSV DNA. For comparison, nonidentical sequences found in ST-FeSV DNA are noted below each numbered line. For ST-FeSV DNA, the *Eco*RI cloning site in the left LTR was aligned with residue 241 of the GA-FeSV DNA sequence. The *Eco*RI cloning site in the right LTR, which was not identical, was aligned with GA-FeSV residue 105. These data suggest that the ST-FeSV LTR contains two *Eco*RI sites and that sequences corresponding to residues 106 through 240 were deleted by cloning. With the exception of this region, approximately 92% of the LTR and leader sequences were conserved between the two strains. A comparable level of homology (95%) was observed between the shared portions of the two FeSV *gag* genes, and an even greater degree of homology (>99%) was found within the common portions of the viral transforming gene, v-*fes* (16a).

The locations of certain characteristic sequences are summarized as follows. (i) A twelve-base-long inverted repeat defined the beginning (residues 1 through 12) and end (residues 471 through 482) of the FeSV LTR sequences. (ii) The adenine plus thymine-rich transcription initiation signal, known as the Goldberg-Hogness or TATA box, was found at residues 311 through 317 with the sequence 5′-TATAAAA. (iii) Twenty-two bases downstream were two overlapping GCG triplets, which are presumed to designate the 5′ capping site for viral RNA (9, 21, 28, 31, 41, 47). (iv) The TATA box was preceded by a characteristic CAT box with the sequence 5′-CCAAT at residues 270 through 274. The relative positions of the CAT, TATA, and GCG sequences corresponded to those expected for eucaryotic RNA polymerase II promotors (12). (v) The hexanucleotide, 5′-AATAAA, typically preceding the polyadenylation sites of eucaryotic genes (29) was found at residues 387 through 392. (vi) This was followed by the dinucleotide CA, 15 to 16 bases downstream, which is presumed to specify the preferred site for polyadenylation. Based on the locations of the 5′ cap triplets and the polyadenylation dinucleotide, the GA-FeSV LTR is composed of 340 nucleotides derived from the 3′ end of viral RNA (U3), followed by R and U5 sequences estimated at 68 and 74 nucleotides, respectively.

After the inverted repeat sequence at the end of the LTR, two T residues and the prolyl tRNA-binding site at residues 483 through 502 mark the beginning of the leader sequence. A consensus splice donor sequence, found at residues 543 to 551, was not preceded by any ATG codons within the upstream sequences corresponding to the extreme 5′ end of the viral RNA. A nine-base deletion relative to GA-FeSV DNA was found within the ST-FeSV leader sequences and was aligned at residues 738 to 746. The leader sequences end at residue 914, after which an ATG codon specifies the p15 amino-terminus of the FeSV polyprotein (16a).

The GA-FeSV LTR sequence was compared with previously published LTR sequences of the Moloney murine sarcoma virus (MSV) (9, 30, 31, 47, 48) and the simian sarcoma virus (SSV) (8) (Fig. 2). A comparison of the GA-FeSV sequences downstream from the CAT box with those of MSV and SSV showed that more than 75% of the GA-FeSV sequences were homologous to nucleotides in the MSV and SSV LTRs. This highly conserved region included the CAT and TATA boxes, the capping triplets, the polyadenylation signals, and major portions of the

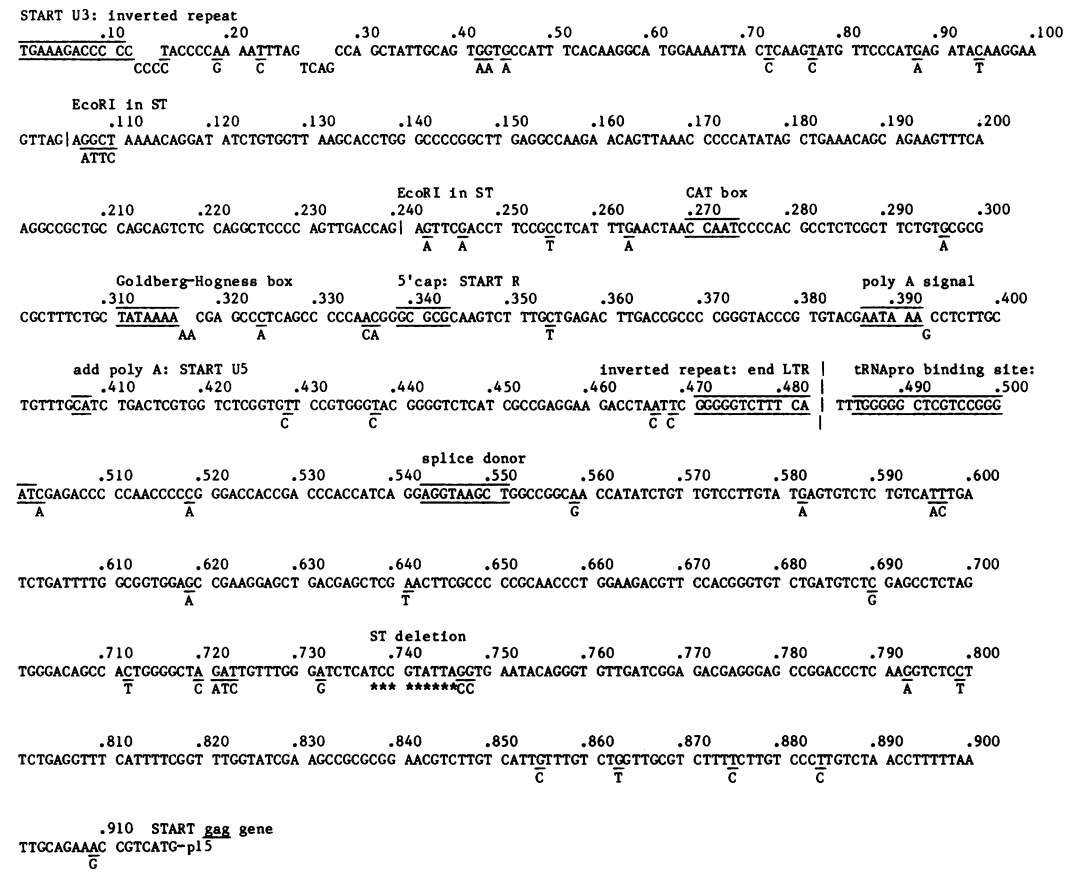

FIG. 1. Nucleotide sequences of the LTRs and 5′ leaders of GA- and ST-FeSV. The junction of the LTR and 5′ leader occurs after residue 482; the A residue at position 915 marks the start of sequences encoding the fusion *gag-fes* polyproteins of each FeSV strain. The numbers above the sequences refer to positions of residues within the GA-FeSV provirus. Base substitutions and additions within ST-FeSV DNA are indicated by residues below the line. The aligned positions of *Eco*RI sites within the ST-FeSV LTR are indicated by the vertical bars after residues 105 and 240. Characteristic sequence configurations discussed in the text are defined by horizontal bars. The asterisks at residues 738 to 746 designate residues present only within GA-FeSV.

inverted repeats. Overlapping hexanucleotide recognition sites for *Sma*I and *Kpn*I were also identified in the R regions of all three viruses, located at residues 369 to 378 in the GA-FeSV LTR sequence.

By aligning the U3 sequences upstream from the CAT boxes, we were able to detect regions of additional homology not previously identified within mammalian retroviral LTRs. Three runs of sequences present in both MSV and SSV appeared to have no counterpart within the FeSV LTR (Fig. 2). These additional residues were positioned between FeSV residues 77–78, 89–90, and 100–101. In addition, the MSV LTR contains a 73-base-pair sequence, of which 69 bases are tandemly and directly repeated within the MSV U3 region (9, 30, 31, 47, 48). The location of the 69-base-pair repeated element was aligned between FeSV residues 100 and 101, whereas the 73-base-pair MSV repeat was aligned with FeSV residues 101 to 163. For the clones analyzed, only a single copy of sequences corresponding to the MSV repeat was found in each LTR. Alignment of the U3 sequences in the manner shown suggested that more than 70% of the GA-FeSV sequences are homologous to nucleotides within MSV and SSV.

The 73-base-pair repeat in MSV has been shown to substitute for the 72-base-pair repeat in simian virus 40 in augmenting the transcription of papovavirus early genes (23). It therefore appears that this region of the MSV LTR serves as an "activator" or "enhancer" element in retroviral RNA transcription. In agreement with

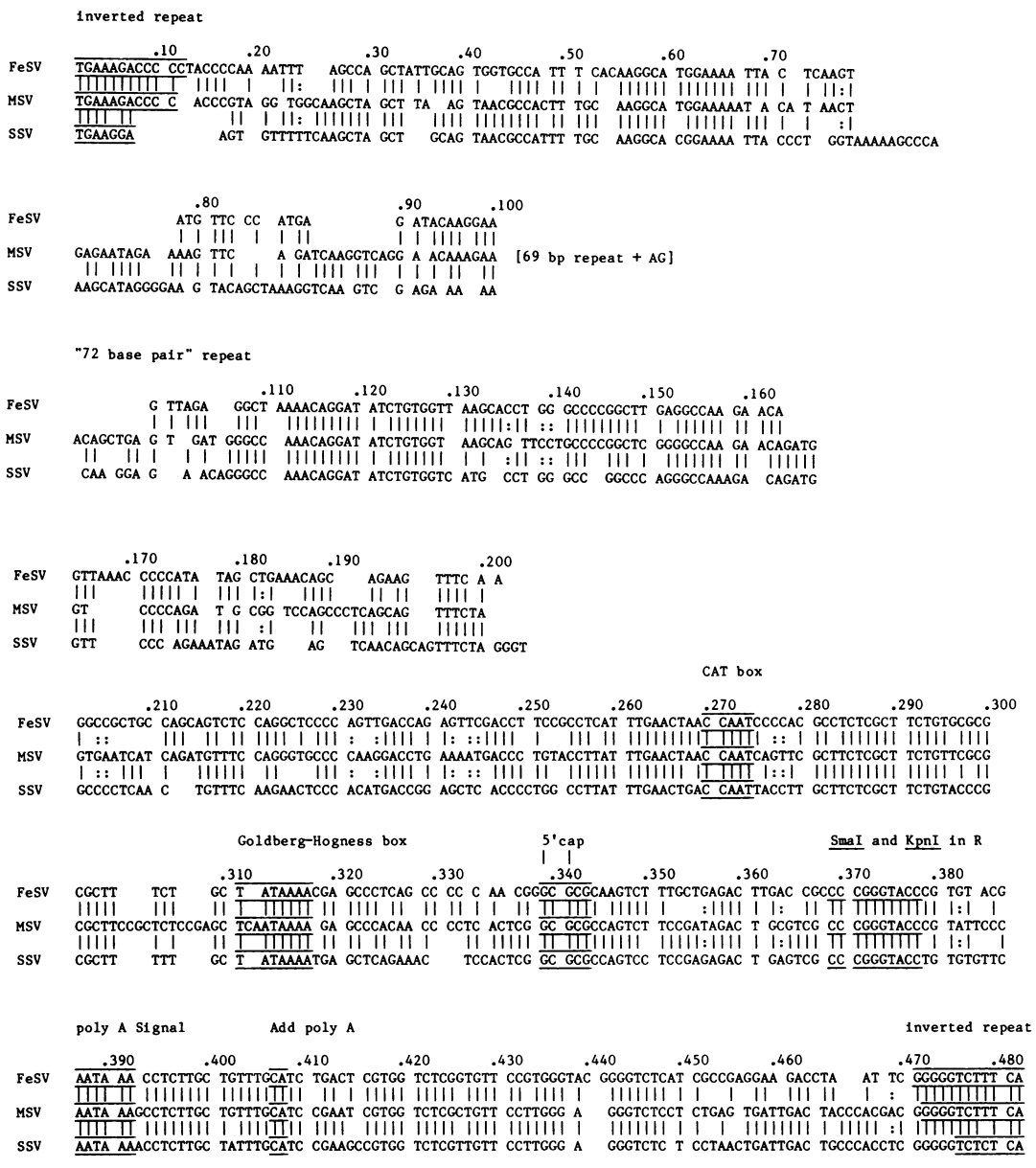

FIG. 2. Comparison of the LTRs of different sarcoma viruses. The GA-FeSV DNA sequence was compared with the published sequences of MSV strain 124 (48) and SSV (8) and was aligned without the assistance of a computer program. The positions of characteristic sequence configurations are noted by horizontal bars, as in Fig. 1. The MSV 69-base-pair repeat occurs after the GAA residues aligned with the FeSV sequence at positions 98 to 100; this is followed by an AG dinucleotide and the MSV 74-base pair repeat, as shown. The latter regions can functionally substitute for the 72-base-pair activator sequence of simian virus 40 (23) (see the text). Homologous restriction sites for *Sma*I and *Kpn*I are underlined (nucleotides 369 through 378).

this interpretation, U3 sequences of murine retroviral LTRs have been shown to activate transcription of cellular proto-oncogenes (3, 6, 7), and have been used to enhance transcription within the mouse mammary tumor virus LTR (G. Hager, personal communication). Comparison of FeSV, MSV, and SSV sequences showed that, although FeSV contains only 63 nucleotides within the predicted region of the 72-base-pair repeat, most of the nucleotides within this region were identical to residues within the other two sarcoma viruses. In particular, FeSV nucle-

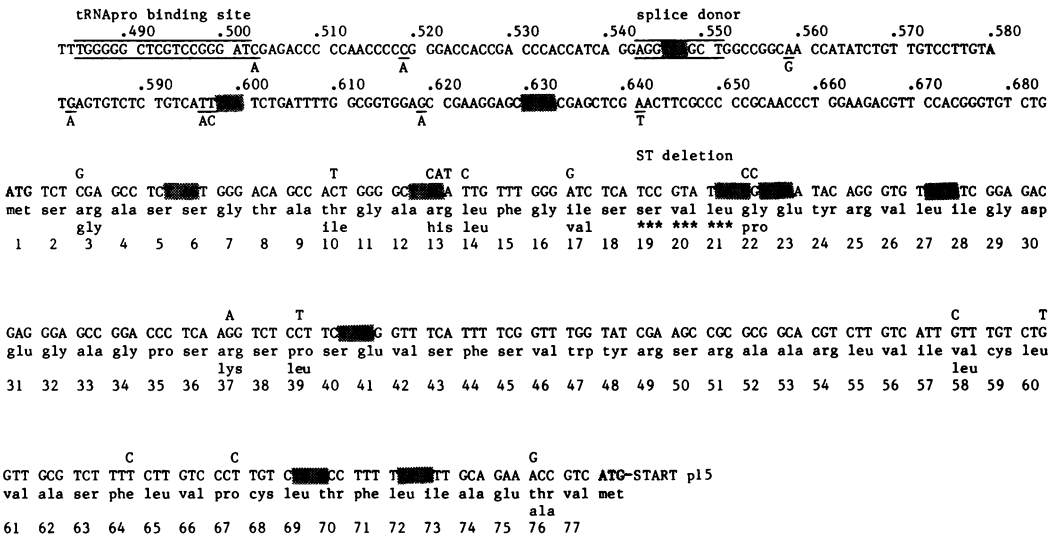

FIG. 3. Translation of the 5′ leader open reading frame of FeSV. The GA-FeSV sequence has been numbered to correspond to the residues indicated in Fig. 1. The prolyl tRNA-binding site and a putative splice donor consensus sequence are indicated by horizontal bars. The positions of potential initiator and terminator triplets are shaded. The ATG codon after residue 683 specifies an amino-terminal methionine, assigned as amino acid no. 1 in the sequence. Amino acid no. 78 is the amino-terminus of the FeSV *gag* gene. Residues 19 through 21 (asterisks) are not represented in ST-FeSV. Other base substitutions within ST-FeSV are indicated as described in the legend to Fig. 1, together with putative changes in amino acid assignments.

otides 112 through 129 showed uninterrupted homology to sequences within both MSV and SSV.

Figure 3 shows the GA- and ST-FeSV leader sequences and designates the positions of potential initiation and termination codons. The GA-FeSV leader contained only three ATG codons. The first, at residues 580 to 582, had undergone a base substitution within ST-FeSV DNA to ATA and was almost immediately followed by a TGA stop codon at residues 597 to 600 in the GA-FeSV sequence. The second ATG codon was followed by a long open reading frame, which could encode 77 amino acids in GA-FeSV (Fig. 3). The nine-base deletion in ST-FeSV DNA maintained the integrity of the reading frame but limited the coding capacity by three amino acids (Fig. 3, residues 19 to 21).

The open reading frame in the FeSV leader sequence was in phase with the ATG codon that specifies the *gag* amino-terminus and could code for a polypeptide of about 7.4 kilodaltons. Amino acid residues 55 through 74 had a high hydrophobicity index (36), contained two cysteines, and were preceded by six amino acids, which included three positively charged arginines at positions 49, 51, and 54. These properties are typical of eucaryotic "signal peptides," which direct nascent polypeptide chains to membranes and are thought to be responsible for the vectorial transport of certain proteins from membrane-bound polyribosomes into the endoplasmic reticulum (4, 5, 25, 27, 33). Indeed, murine leukemia viruses encode two forms of the *gag* gene precursor (11, 13, 34). The larger of the two contains about 7 kilodaltons of additional amino-terminal residues and is glycosylated, suggesting that the presence of signal sequences leads to compartmentalization of this form of the *gag* precursor within the endoplasmic reticulum and its subsequent glycosylation (34, 35). The glycosylated *gag* gene products are transported to the plasma membranes of infected cells but are not processed into virions (22, 43).

Unlike murine sarcoma viruses, which lack the signal sequences (30, 48) and do not encode glycosylated *gag* gene products, glycosylated forms of both the ST- and GA-FeSV polyproteins have been detected in FeSV transformants (40). The two ATG codons may be able to initiate translation of glycosylated and nonglycosylated forms, respectively, of the polyprotein, from full-genome-length mRNA molecules. Alternatively, full-length transcripts might be spliced, removing the upstream ATG codon and eliminating translation of glycosylated products; a potential acceptor sequence between the two ATG codons starts at residue 795 (TCTCCTTCTGAG↓GT).

Although the leader sequences corresponding to the FeSV open reading frame did not show any clear homology to sequences in MSV, virtu-

ally all of the noncoding leader sequences were homologous to MSV sequences (data not shown). Together with the relationship of sequences within the LTR regions of these genomes, the results suggest that the helper virus-derived regions of these different sarcoma virus isolates may have originated from a common sequence. Previous comparisons of the viral structural genes and their products are consistent with the possibility that FeSV and SSV were independently derived after interspecies transmission of ancestral rodent retroviruses to cats and primates, respectively (1, 2, 24). It appears, then, that noncoding regulatory sequences at the termini of certain retroviral RNA genomes have been as highly conserved as the viral structural genes.

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