Nucleotide Sequence of the Genes for the Simian Virus 40 Proteins VP2 and VP3*

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We have determined the nucleotide sequence of the DNA of simian virus 40. The preceding report (Dhar, R., Reddy, V. B., and Weissman, S. M. (1978) J. Biol. Chem. 253, 612-620) presents the sequence of a portion of the simian virus 40 DNA that overlaps the region encoding the 5' end of the minor structural protein VP2. We report here the sequence of the remainder of the genes for the minor structural proteins VP2 and VP3. The results indicate that the mRNA for the two proteins is read in the same phase and the initiation site for VP3 lies within the structural gene of VP2. The codons of the COOH-terminal amino acids of VP2 and VP3 are read in a second phase as the codons of the NH2-terminal amino acids of VP1.

See the preceding paper (1) for the background of these studies.

METHODS

These have been described in the preceding publication (1).

RESULTS

The restriction endonuclease cleavage pattern of this region of SV40 DNA is presented in the accompanying publication (1). Part of the nucleotide sequence of the fragment Hae III-I has been approach by separately by RNA and DNA analytic methods. We are grateful to Dr. J. Lautenberger (University of North Carolina) who drew our attention to the presence of errors in an earlier version of this sequence. A representative oligonucleotide map of a T, RNase digest of an [α-32P]ATP labeled transcript of fragment Hae I is shown in Fig. 1 and Table I.

Much of the sequence of the remainder of HindII,III-D, -E, and -K could be obtained by analysis of products of limited venom diesterase digestion of DNA fragments labeled with 32P at the 5' termini. Such preparations were made for all the available restriction fragments within this region. Representative results are shown in Figs. 2 to 7. (The sequence read on the maps follows the mobility shift data presented by Tu Chen Pei et al. (2.) These results were confirmed and the sequence was extended by examination of products of limited

FIG. 1. Products of extensive T, RNase digestion of the transcript of Hae III-I. The DNA fragment Hae III-I was transcribed with Escherichia coli RNA polymerase in the presence of [α-32P]ATP. The radioactive RNA was then digested with T, RNase and the resulting oligonucleotides were fractionated by electrophoresis from left to right on Cellogel at pH 3.5 and by chromatography from below upward on a DEAE-cellulose thin layer chromatographic plate (see "Methods"). The numbers refer to oligonucleotides whose sequence is shown in Table I. Products not labeled by [α-32P]ATP are not shown in the chromatogram.

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The abbreviations used are: SV40, simian virus 40; Hae II,III, enzymes from Hemophilus influenzae, strain d; Alu I, enzyme from Arthrobacter luteus; Eco RI,II, enzymes from plasmid-bearing Escherichia coli.
FIG. 2 (left). Products of partial snake venom diesterase digestion of the DNA fragment Hae III-H labeled at the end within HindII,III-D. The oligonucleotides were fractionated as in Fig. 1. Letters indicate residues removed to generate successively shorter products.

FIG. 3 (center). Products of partial snake venom diesterase digestion of the DNA fragment Hae III-I labeled at the end nearest HindII,III-E.

FIG. 4 (right). Products of partial snake venom diesterase digestion of the DNA fragment Hae III-J labeled at the end nearest HindII,III-D.

FIG. 5 (left). Products of partial snake venom diesterase digestion of the DNA fragment HindII,III-E labeled at the end nearest HindII,III-K.

FIG. 6 (center). Product of partial snake venom diesterase digestion of the fragment HindII,III-D labeled at the end nearest HindII,III-E.

FIG. 7 (right). Product of partial venom diesterase digestion of the fragment Alu I-P labeled at the end within Hae III-I.
TABLE I

| Oligonucleotide Number | Sequence | Strand from which oligonucleotide was transcribed |
|------------------------|----------|--------------------------------------------------|
| 1                      | CCUCCACAUUUAAGCUCAACUCCUG | E |
| 2                      | CUCCUCAUUAAUACAAG          | E |
| 3                      | AACAACUGCGCUCCUG           | E |
| 4                      | CACAUCAUGCUCCACAG          | E |
| 5                      | CAACAUAAAG                 | L |
| 6                      | CAGCAUG                    | E |
| 7                      | AAAASACUG + UCACCCUG       | L+E |
| 8                      | AAACAUUU (U)               | L |
| 9                      | AACCUUUGU                 | L |
| 10                     | UCCCUUGU                  | E |
| 11                     | UCAGCAUGU                 | L |
| 12                     | UCACACUGU                 | L |
| 13                     | CUCCUUGU                  | L |
| 14                     | CCCAACUGU                 | L |
| 15                     | AAACUGU                   | L |
| 16                     | UCCUUGU                   | L |
| 17                     | UCACACUGU                 | L |
| 18                     | CCUCUGU                   | L |
| 19                     | UCACACUGU                 | L |
| 20                     | CCUCACAGU                 | L |
| 21                     | UCAGCAUGU                 | L |
| 22                     | CCACACUGU                 | L |
| 23                     | CCACACUGU                 | L |
| 24                     | AACACUGU                  | L |
| 25                     | AAACUGU                   | L |
| 26                     | UCACACUGU                 | L |
| 27                     | UCACACUGU                 | L |
| 28                     | UCACACUGU                 | L |
| 29                     | UCACACUGU                 | L |
| 30                     | UCACACUGU                 | L |
| 31                     | UCACACUGU                 | L |
| 32                     | UCACACUGU                 | L |
| 33                     | UCACACUGU                 | L |
| 34                     | UCACACUGU                 | L |
| 35                     | UCACACUGU                 | L |
| 36                     | UCACACUGU                 | L |
| 37                     | UCACACUGU                 | L |
| 38                     | UCACACUGU                 | L |
| 39                     | UCACACUGU                 | L |
| 40                     | UCACACUGU                 | L |
| 41                     | UCACACUGU                 | L |
| 42                     | UCACACUGU                 | L |
| 43                     | UCACACUGU                 | L |
| 44                     | UCACACUGU                 | L |
| 45                     | UCACACUGU                 | L |
| 46                     | UCACACUGU                 | L |
| 47                     | UCACACUGU                 | L |
| 48                     | UCACACUGU                 | L |

(a) See Fig. 1 of preceding report.
(b) See Fig. 1. Oligonucleotides predicted by the sequence but not labeled with P-32 ATP are not shown but were detected in comparative proportions labeled with other radioactive nucleotides.
(c) Sequence of each oligonucleotide was deduced by digestion with pancreatic or U2 RNase, in comparison with complementary strand sequences and by comparison with DNA sequences.
(d) "L" and "E" refer to the DNA strand whose respective transcripts include sequences of SV40 late mRNA or its complement.

Fig. 8. Products of limited chemical degradation of the DNA fragment Hae III-H. The DNA fragment was divided into four aliquots and processed by the procedure of Maxam and Gilbert (3). Electrophoresis was from above downward in a 20% acrylamide gel. The patterns are representative of multiple patterns obtained after varying times of electrophoresis. The A, AG, CT, and C above the columns refer to products generated by selective cleavage of DNA of the bases A, A, G, C, and T, or C. Letters to the right of the autoradiogram indicate successive residues in the sequence. a, label at start of fragment nearest Hae III-J; b, label at end of fragment nearest Hae III-I.

The sequence of the region of SV40 DNA through the entire gene for VP2 and VP3 and the codons for the NH2-terminal amino acids of VP1 is shown in Fig. 13.

Several preparations of radioactive RNA were obtained from cells that had been infected for 36 to 48 h with SV40 virus. This RNA was extracted from disrupted cells after the

* W. Beattie, T. Kempe, S. M. Weissman, and W. Konigsberg, unpublished observations.
nuclei had been removed by centrifugation and was further fractionated on oligo(dT)-cellulose. The retained RNA was annealed to restriction fragments \(Hae\text{ III-I}\) (Fig. 1) or \(Eco\text{ RI}\)-I-E, eluted, and digested with \(T_1\) RNase. The products of \(T_1\) RNase digestion of the mRNA were further analyzed by digestion with pancreatic RNase. The oligonucleotides obtained were always those predicted from the sequences of the fragment even though one end of this fragment lies within about 40 nucleotides of the initiation codon for VP1 and the 16 S late mRNA that directs the synthesis of VP1 has been reported to have its 5' end near the initiation codon site (5).

**DISCUSSION**

The nucleotide sequence of this region of SV40 DNA (Fig. 13) shows that the late strand RNA transcript contains an AUG triplet beginning within \(Hind\text{ III-D}\) fragment 41 nucleotides from the \(Hind\text{ III-L-Hind\text{ III-D}}\) junction and that this AUG is followed in phase by 352 triplets extending across the remainder of the \(Hind\text{-D fragment}\), all of the \(Hind\text{ III-E fragment}\) and into the \(Hind\text{ III-K fragment}\), terminated by a UAA codon in the \(Hind\text{-K fragment}\), 126 nucleotides from the \(Hind\text{-E-Hind\text{-K}}\) junction. No other reading frame of the late strand transcript of \(Hind\text{ III-D or Hind\text{ III-E}}\) can be translated into a large peptide. The SV40 fragments \(Hind\text{ III-D}\) and \(Hind\text{ III-E}\) include the regions where temperature-sensitive mutants of Group D have been mapped by marker rescue experiments and complementation studies with deletion mutants of SV40 (6-8). The 5' end of large, late mRNA has been located by electron microscopy and by nucleic acid sequence studies near the \(Hind\text{ III-C-Hind\text{ III-D}}\) junc-
tion (5, 9), probably between 30 and 10 nucleotides upstream from the AUG initiating the long run of in-phase sense codons. The large, late RNA directs the synthesis of VP2 in cell-free protein-synthesizing systems (10). With polyoma, a revertant of a temperature-sensitive mutant virus simultaneously changes a peptide in VP2 and the same peptide in VP3.3

The evidence in aggregate indicates almost certainly that this region of DNA includes the codons for VP2 and VP3.

The sequence is consistent with the suggestion that VP3 is read from codons from DNA that encodes a portion of VP2 and VP3 and is translated in the same phase as VP2. The estimated molecular weight of VP2 from sodium dodecyl sulfate-gel electrophoresis is 37,000 to 39,000. The maximum number of amino acid residues expected from the DNA sequence is 352, so that the molecular weight by sodium dodecyl sulfate gel estimation is just slightly larger than that predicted. Both VP2 and VP3 synthesized in vivo are reported to have blocked NH₂ termini4 so that it is not known whether they retain the terminal methionine. In cells infected with polyoma virus, pulse label experiments and studies with inhibitors of proteolysis have not given any evidence that VP3 is derived by cleavage of VP2.5 With the related virus, polyoma, there is some evidence that polyoma VP2 may be translated from an 18 S message somewhat smaller than the 19 S message which directs the synthesis of VP2.6 One hundred and eighteen codons in phase downstream from the AUG that is the probable initiator for VP2 synthesis is the next methionine codon to appear within the sequence. It

3 Gibson, W., Hunter, T., Cogen, B., and Eckhart, W. (1977) J. Virol., in press.

4 Gibson, W., and Hunter, T., personal communication.

5 T. Hunt and G. Wade, personal communication.

6 A. Smith, personal communication.
appears quite possible that this codon actually acts as initiator codon for the synthesis of VP3, perhaps from a shorter messenger RNA. Following this methionine, there are 234 codons in phase. The estimated molecular weight of VP3 from sodium dodecyl sulfate-gel electrophoresis is 27,000, so that there is fairly good agreement between the predicted amino acid sequence from the DNA data and the estimated molecular weight of the protein.

The amino acid analysis of gel-purified VP3 has been reported. Comparison of the reported amino acid composition (11) with that calculated from the codons that we believe direct the synthesis of VP3 shows some similarities and differences. Direct analyses of the protein showed no cystine in VP3. The nucleotide sequence also shows no cystine codons in either VP2 or VP3. The most striking discrepancy between predicted and observed amino acid content for VP3 is a reported value of 18% for glycine residues, in contrast with our estimate of approximately 7%. The valine content estimated at approximately 5% is substantially lower than the reported value of 6.9%. A complete comparison is shown in Table III. Presumably, at least some of the residual differences are due to impurities in the VP3 isolated from sodium dodecyl sulfate gels, although the possibility for genetic variation between virus strains or even the presence of other material associated with VP3 cannot be ruled out.

Although no function is known for VP2 during infection or in virus structure, the existence of regions of the genome coding only for VP2 indicate that it plays some specific role. The NH₂-terminal segment of VP2, outside of VP3, has few basic amino acid residues and the content of hydrophobic amino acids is nearly as high as that in the NH₂-terminal peptides of proteins thought to be associated with cell membranes (12). VP2 may be involved in virus membrane interaction during virus assembly or cell penetration. The COOH-
Fig. 12. Products of limited chemical degradation of the fragment HindIII,E labeled at the end nearest HindIII,D. The autoradiogram is labeled as in Fig. 8. a, short electrophoretic run; b, long electrophoretic run.

The overlap of the codons for the 3' end of VP2 and VP3 with the codons that are read in another phase as part of the VP1 gene is the first example in which interpenetrating genes function in eukaryotic cells. It is much less extensive than the overlapping of genes recently described by Sanger et al. for φX174 (13). The total size of the genome of SV40 is very similar to that of φX174 and SV40 consumes a larger portion of its sequence information in nontranslated regions than does the bacteriophage. It is therefore not unexpected that mechanisms for maximization of utilization of genetic information do occur. Production of overlapping genes translated in the same phase is an alternative to having interpenetrating genes in which the codons are translated into two different phases.

The structures around the 5' ends of the VP3 message are of some interest. Beginning within about 80 nucleotides upstream from the AUG initiation codon for VP3 is the sequence UUUUUUA in the VP2 mRNA. The sequence UUUUUU purine has been recognized as a part of a common termination signal for a variety of prokaryotic transcripts (14). It also occurs as one of the potential termination signals for the VA-RNA synthesized in adenovirus-infected human cells (15). The possibility that this or similar sequences could function as transcription termination sites in SV40 has been discussed elsewhere (16). If this sequence did act as a transcription terminus of VP3 has a cluster of 5 and another of 6 basic amino acids, somewhat reminiscent of the clusters of basic amino acids in histones and these regions of the proteins might make contact with DNA, perhaps substituting for histone H1.
Sequence of Coding Region of SV40 VP2 and VP3 mRNA

FIG. 13. Nucleotide sequence of the mRNA for VP2 and VP3. Nucleotides are grouped in triplets on the phase in which VP2 and VP3 are translated. Above each triplet is written the three letter code of the corresponding amino acid. The sequence extends across most of HindIII-D, all of HindIII-E, and a part of HindIII-K.
arrest or termination site, there might be a mechanism by which transcription arrest shortly upstream from the 5' end of the VP3 coding region would permit the formation of an mRNA whose 5' end lay near the coding region for VP3 and which would code for VP3 but not VP2. Preceding the putative termination site, there might be a mechanism influencing the relative amounts of VP2 and VP3 made during the later phases of infection.

Very recently the nucleotide sequences have become available for human (20) and rabbit (21) \( \beta \)-mRNA and partial sequences for human \( \alpha \)-globin mRNA. These sequences show a bias in the utilization of synonym codons. For example, UUG is not used at all in the \( \alpha \)-chain mRNA and either or both of these alternatives or a more complex process might generate the VP2 and VP3 mRNA. The availability of large amounts of SV40 DNA of known structure should provide a favorable system for probing fidelity of RNA transcription or processing with animal cell enzymes. If this or a similar interpretation of the sequence is correct, there is probably a mechanism influencing the relative amounts of VP2 and VP3 made during the later phases of infection.

TABLE II

| Oligonucleotide | Sequence [c] |
|-----------------|-------------|
| 2               | CAAAUUUAG   |
| 3               | CAAUUCAAG   |
| 4               | CAAACUCAAAG|
| 5               | AAAAUUUG    |
| 6               | AAAUUUUG    |
| 7               | CUCUUACUAAG|
| 8               | CUCUCUAG    |
| 9               | CUCAAG      |
| 10              | UCUCUAG     |
| 11              | UACUG       |
| 12              | ACCUG       |
| 13              | UCAG        |
| 14              | UACUG       |
| 15              | CCAACAG     |
| 16              | CCAAG       |
| 17              | ACAAG       |
| 18              | AAG         |
| 19              | ACAG, CAG   |
| 20              | CAG         |
| 21              | UUG         |
| 22              | UCUUCAAGUUG|

(a) See Footnote (d), Table I.
(b) See Fig. 1.
(c) See Footnote (c), Table I.
(d) This oligonucleotide is not seen in transcripts of \( \text{Hae III-I} \) prepared in vitro since its 5' terminal nucleotide is the 5' terminus of the L strand of \( \text{Hae III-I} \).
(e) This oligonucleotide is not seen in Fig. 1 since it is not labeled with \( <^{32} \text{P} \). ATP.
(f) This oligonucleotide comes from the sequence of DNA fragment \( \text{Hae III-II} \) adjacent to \( \text{Hae III-I} \).

TABLE III

| VP2 outside of VP3 | VP3 | Reported values from amino acid analyses |
|-------------------|-----|----------------------------------------|
| Residues Moles per cent | Residues Moles per cent | VP3 | VP1 |
| Alanine 29 24.8 | 16 6.8 | 6.6 | 6.4 |
| Arginine 0 0 | 21 9.0 | 5.7 | 3.8 |
| Asparagine 0 0 | 11 4.7 | 7.9 | 10.6 |
| Aspartic acid 3 2.6 | 12 5.1 |  |
| Cystine 0 0 | 0 0 |  |
| Glutamine 6 5.1 | 17 7.3 | 11.5 | 11.5 |
| Glnamic acid 5 5.1 | 12 5.1 |  |
| Glycine 12 10.3 | 12 5.1 | 18.2 | 9.7 |
| Histidine 1 1.9 | 4 1.7 | 1.4 | 1.4 |
| Isoleucine 8 6.8 | 10 4.3 | 3.2 | 4.1 |
| Leucine 12 10.3 | 18 7.7 | 8.6 | 9.1 |
| Lysine 1 1.9 | 9 4.3 | 5.2 | 6.5 |
| Methionine 1 1.0 | 2 1.3 | 1.9 | 1.8 |
| Phenylalanine 3 2.6 | 8 3.4 | 3.2 | 3.7 |
| Proline 3 2.6 | 14 6.0 | 5.2 | 6.5 |
| Serine 7 6.0 | 22 9.4 | 7.1 | 5.7 |
| Threonine 9 7.7 | 15 6.4 | 4.2 | 7.6 |
| Tyrosine 3 2.6 | 11 4.7 | 3.1 | 2.6 |
| Valine 13 11.1 | 19 5.1 | 6.9 | 9.9 |

(a) See Fig. 13.
(b) Moles/100 mol of all amino acids including tryptophan.
(c) Moles/100 mol of amino acids other than tryptophan. Data from Ref. 12.

Either or both of these alternatives or a more complex process might generate the VP2 and VP3 mRNA. The availability of large amounts of SV40 DNA of known structure should provide a favorable system for probing fidelity of RNA transcription or processing with animal cell enzymes. If this or a similar interpretation of the sequence is correct, there is probably a mechanism influencing the relative amounts of VP2 and VP3 made during the later phases of infection.

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TABLE IV

| Residue | Moles/100 mol of all amino acids including tryptophan |
|---------|------------------------------------------------------|
| Alanine | 29                                                   |
| Arginine| 0                                                    |
| Asparagine | 0                                               |
| Aspartic acid | 3                                                |
| Cystine | 0                                                    |
| Glutamine | 6                                                |
| Glnamic acid | 5                                             |
| Glycine | 12                                                   |
| Histidine| 1                                                     |
| Isoleucine | 8                                               |
| Leucine | 12                                                   |
| Lysine | 1                                                     |
| Methionine | 1                                               |
| Phenylalanine | 3                                               |
| Proline | 3                                                    |
| Serine | 7                                                     |
| Threonine | 9                                                 |
| Tyrosine | 3                                                    |
| Valine | 13                                                    |

(a) J. T. Wilson, C. A. Marotta, B. Forget, and S. M. Weissman, unpublished results.
### TABLE IV
Codon usage in mRNA for SV40 structural proteins VP2 and VP3

| 1st position | 2nd position | 3rd position |
|--------------|--------------|--------------|
|              | C | A | G | U |
| VP2 outside of VP3 gene | C | 0 | 1 | 0 | 1 |
|              | A | 2 | 5 | 0 | 2 |
|              | G | 0 | 1 | 0 | 2 |
|              | U | 1 | 0 | 1 | 1 |
|              | C | 1 | 0 | 1 | 0 |
|              | A | 2 | 1 | 1 | 4 |
|              | G | 0 | 0 | 0 | 1 |
|              | U | 6 | 0 | 1 | 4 |
|              | C | 3 | 1 | 2 | 0 |
| VP2 codons overlapping VP1 codons | G | 4 | 4 | 4 | 1 |
|              | A | 0 | 2 | 3 | 6 |
|              | C | 23 | 1 | 3 | 5 |
|              | U | 0 | 0 | 0 | 0 |
|              | C | 0 | 0 | 0 | 0 |
|              | A | 1 | 0 | 0 | 3 |
|              | G | 0 | 0 | 0 | 1 |
|              | U | 5 | 3 | 0 | 4 |
| VP3 outside of VP1 gene | C | 2 | 2 | 0 | 1 |
|              | A | 2 | 11 | 0 | 2 |
|              | G | 0 | 5 | 0 | 1 |
|              | U | 9 | 1 | 1 | 3 |
|              | C | 4 | 2 | 1 | 0 |
|              | A | 3 | 2 | 6 | 2 |
|              | G | 0 | 0 | 7 | 3 |
|              | U | 6 | 7 | 5 | 8 |
|              | C | 2 | 5 | 1 | 0 |
|              | G | 3 | 8 | 2 | 6 |
|              | A | 0 | 4 | 2 | 2 |
|              | U | 8 | 6 | 4 | 4 |
|              | C | 0 | 3 | 0 | 0 |
|              | U | 3 | 0 | 0 | 5 |
|              | G | 0 | 0 | 6 | 5 |
|              | U | 5 | 8 | 0 | 8 |

leucine are the UU purine codons. Except for the region of overlap of VP2 and VP1 genes, there is a 21/2-fold excess of uridylic acids, as compared with cytidylic acids in third position of termination codons. This is reminiscent of the excess of uridylic acids noted in dX174 and also in the segment of the SV40 early mRNA whose sequence has been reported by Volckaert et al. (22). The reasons for such selection are unknown. They could reside in requirements of the structure of DNA itself, in preference for the GU wobble pair in the third position of codons as compared with the GC pair, or in preference for tRNAs which contain adenyllic acid or modified uridylic acid in the first position at the anti-codon and which would pair preferentially with codons having uridylic acid in third position. The results indicate that the pattern of codon utilization in hemoglobin mRNA is not a general feature of animal cell mRNA.

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