

edly exist, as illustrated by the discovery of either isoleucine or threonine at position 75 in human $\Lambda\gamma$ -globin (19). Also a silent polymorphism involving a neutral amino acid substitution (valine or isoleucine) at residue 31 of human skeletal muscle carbonic anhydrase (CA III) has been reported (20); this was detected by reverse-phase HPLC separation of tryptic peptides of CA III from pooled muscle samples. Silent substitutions in hemoglobin may also be common in the baboon, as illustrated by the description of hemoglobin Papio B (4).

The ability to detect amino acid substitutions in proteins has provided a biochemical means of estimating the amount of genetic variation in human populations and the frequency of mutations (21). The screening procedures used for these estimates include electrophoresis and measurements of enzyme activity and protein stability. The discovery of hemoglobin Beirut illustrates the potential of reverse-phase HPLC for the detection of neutral amino acid substitutions in proteins. Since the frequency of neutral amino acid substitutions could be two to three times greater than the frequency of charge substitutions, reverse-phase HPLC increases our ability to detect genetic variations in proteins and to estimate the frequency with which mutation results in a previously cryptic type of protein variation.

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18. Other neutral amino acids at residue 126 in β or β -like chains [see (16)] are leucine (spider monkey, horse, llama, pig, and frog β chain; sheep and cow β -like and γ -like chains; and goat β -like

chain); threonine (kangaroo β chain); cysteine (chicken β chain); and methionine (human δ chain).

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23. Supported in part by NIH grant R01-HL25541. We thank J. V. Neel, D. Rucknagel, and D. Hewett-Emmett for helpful suggestions in the preparation of this manuscript.

10 February 1983

An Inverted Repeat Borders a Fivefold Amplification in Satellite DNA

Abstract. One variant of a complex satellite DNA of the Bermuda land crab is significantly longer than the average repeat unit of the satellite. The extra DNA in the variant is accounted for by a fivefold tandem amplification of a 0.142-kilobase sequence. The amplified sequence is bounded by a tetranucleotide inverted repeat; the upstream arm of the inverted repeat is missing from two other variants of the satellite. The latter variants contain only one copy of a sequence that is closely related to the amplified sequence. By contrast, in several satellite DNA's of other organisms, extra DNA is inserted.

Bermuda land crab DNA contains two satellites. One of them has a guanine and cytosine (G+C) content of 63 percent, a density in CsCl gradients of 1.721 g/cm³, and comprises 3 percent of the total DNA (1). When products of an Eco RI

digest of that satellite were inserted into the Eco RI site of plasmid pBR322 and amplified in *Escherichia coli* strain HB101, 13 of the 16 clones recovered had inserts 2.06 to 2.09 kilobases (kb) long, similar to the sizes of repeat units

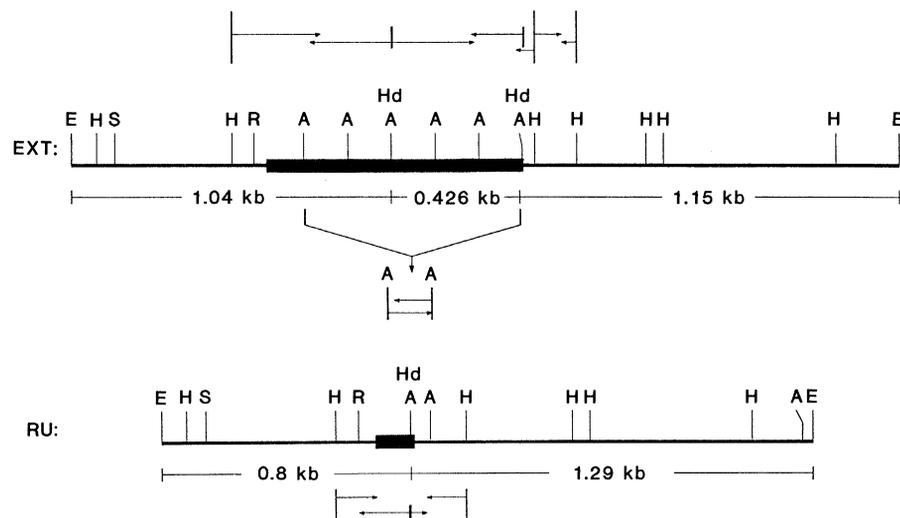


Fig. 1. Restriction maps of EXT and RU showing sites for Alu I (A), Eco RI (E), Hind III (Hd), Hinf I (H), Rsa I (R), and Sau3A I (S). The amplified (six copies) of the 0.142-kb repeats in EXT and the homologous 0.127-kb sequence in RU are indicated by filled boxes. The Hind III fragments seen in Fig. 2, A and B, are shown below each map. Fragments were sequenced by the Maxam-Gilbert procedure (7). (i) Sequencing strategies for the Hinf I 0.989-kb and the 0.136-kb fragments of EXT are shown above its restriction map; the strategy for a mixture of five 0.142-kb repeats resulting from digestion of the 0.989-kb fragment with Alu I is below. (ii) One of several sequencing strategies for Hinf I fragments of RU that contain the homolog to the amplified sequence is shown below the restriction map of RU; sequencing was also done with Hpa II fragments (5). Vertical bars that terminate arrows indicate the 5' ends of fragments that were labeled with ³²P; the length of arrows indicates the distance read. Labeled fragments were either strand-separated or cut with restriction enzymes at the sites shown.

recovered from digests of cellular satellite (2). The sizes of three other cloned inserts deviated from that of the major repeat unit: (i) an extended variant (EXT) of ~ 2.65 kb, (ii) a truncated variant (TRU) of ~ 1.69 kb, and (iii) a dimer of ~ 4.14 kb.

Extra sequences in other very highly repeated (VHR) DNA's from rye (3) and calf (4) are unrelated to the VHR DNA and are thought to be inserted. By contrast, we present evidence that the extra DNA in the extended cloned variant EXT of land crab satellite is an amplification of a single copy of a 142-base pair (bp) sequence closely related to a sequence of 128 ± 2 bp found in the other two variants.

Restriction maps of EXT and one of the cloned variants having the size of the major repeat unit (RU; 2.089 kb) show considerable homology (Fig. 1). Not identified here is a 0.108-kb deletion from EXT (5). A series of Alu I sites uniformly spaced at intervals of 0.142 kb in EXT are absent from both RU and TRU, suggesting that the extra DNA in EXT is localized at a single site.

Hind III cuts EXT into fragments of 1.04, 0.426, and ~ 1.15 kb, respectively, from the 5' end (lane 2 in Fig. 2A). A ³²P-labeled probe of the Hind III 0.426-kb fragment from the region of extra DNA in EXT hybridizes with fragments from RU and TRU (6), an indication that sequences homologous to the extra DNA are localized in a 0.8-kb and a 0.79-kb fragment upstream from the single Hind III site in RU and TRU, respectively. That probe also hybridizes with all clones containing the Eco RI inserts described above as well as to the band 2.07 ± 0.1 kb long resulting from digestion of native satellite with Eco RI (data not shown). Removal of a 0.168-kb fragment from the 3' end of the 0.8-kb RU fragment by digestion with Rsa I almost totally suppressed hybridization (lanes 5 and 6 in Fig. 2B).

We sequenced (Fig. 1) (5, 7) fragments that contained the amplified sequences in EXT or their homologs from RU and TRU. The sequences (Fig. 3) confirm that the Hinf I 0.989-kb fragment in EXT is the site of the extra DNA and that the extra DNA is a fivefold amplification of a 0.142-kb sequence that results in six tandem copies.

The RU and TRU sequences that are homologous to the 0.142-kb amplified sequence in EXT each contain two deletions at the same sites (Fig. 3A), which shorten them to 127 and 130 bp, respectively. The homology between the amplified sequence and its counterparts in RU and TRU is 83.1 and 83.8 percent, re-

spectively; the homology of the latter two segments to each other is 95 percent. Immediately upstream from the beginning of the amplified sequence, 24 bases present in RU and TRU are deleted from EXT. For 105 bases further upstream and for 173 bases downstream from the amplified sequence, the homology among all three variants is greater than 90 percent (Fig. 3A). The homology between RU and TRU throughout these same regions is greater than 97 percent.

Between bases 94 and 193 in the Hind III 0.989-kb fragment of EXT (including the first 88 bases of the 142-bp repeat), 48 percent of the bases in one reading frame (allowing for an insertion in one triplet AAG to AAAG at position 127)

form the triplets AAG or AAC (AAG/C). Another 45 percent are a single base modification of AAG/C. Direct repeats and reverse repeats of up to 16 bases and more than 80 percent homology are frequent in this region (bracketed sequences in Fig. 3A). However, these appear to reflect the high frequency of AAG/C and its modifications and not a higher degree of organization. The deletions preceding and within the 0.142-kb repeat in EXT or the homologous sequence in RU and TRU occur within the region rich in AAG/C triplets. That all deletions are multiples of 3 is consistent with the model of deletion formation by slipped mispairing (8-10) of repeats of the frequently occurring trimer.

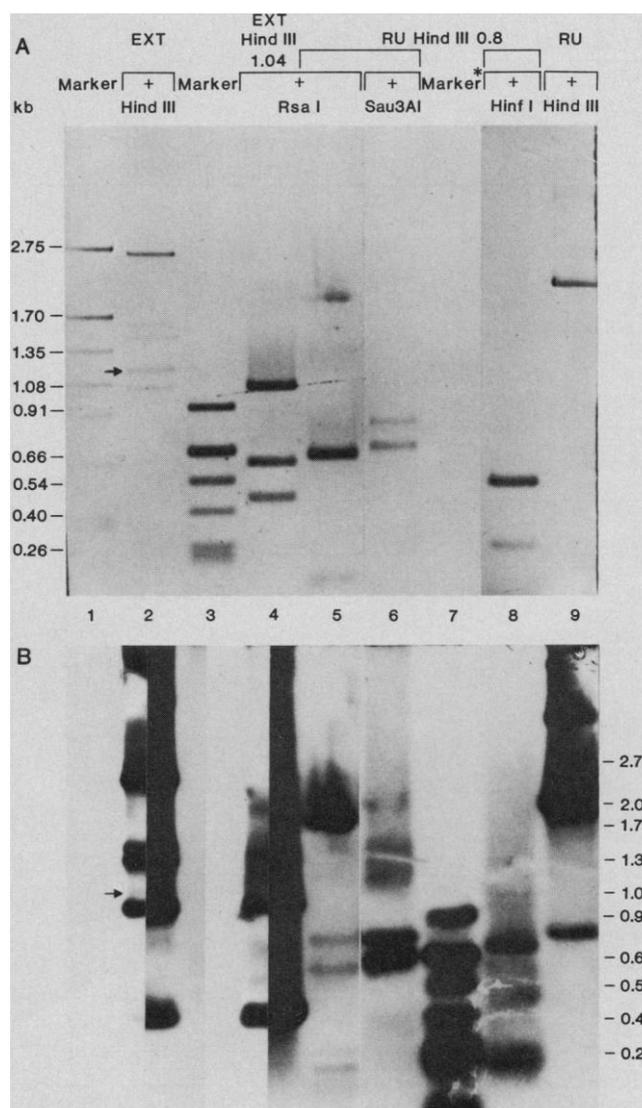


Fig. 2 Localization of the sequence in RU homologous to the amplified sequence in EXT. (A) Agarose gel (1.5 percent) with 1 μ g of DNA stained with ethidium bromide in each lane. (Lanes 2 and 4) EXT; (lane 9) RU; (lanes 5, 6, and 8) 0.8-kb Hind III fragment from RU; restriction enzymes used for digestions are indicated. (B) Autoradiogram of nitrocellulose filter after transfer of DNA from gel in (A) and hybridization (11) with probe of nick-translated Hind III 0.426-kb fragment from EXT. In lanes 2, 3, and 4 of (B), paired half-lanes of underexposed and overexposed photographs are used in order to resolve bands of either very faint or intense hybridization. Strong hybridization occurs with fragments from EXT containing three copies of the amplified sequence (lane 2, 0.426 kb and lane 4, 0.44 kb) and with RU fragments containing a single copy of the homolog to the amplified sequence (lane 6, 0.66 kb; lane 8, 0.25 kb; and lane 9, 0.8 kb). Inefficient binding to the nitrocellulose filter of a 0.168-kb fragment (lane 5) containing one copy

of the homolog may account for the weak hybridization seen. Other strong signals are to undigested and partially digested fragments containing the amplified sequence or its homologs. The specificity of hybridization is attested to by the lack of hybridization with marker DNA's (lanes 1 and 3) or with a large fragment downstream from the Hind III site [arrows, lane 2 in (A) and (B)]. Nevertheless, faint hybridization occurs with several fragments from the region upstream to the amplified DNA (lane 4, 0.61 kb; lane 5, 0.63 kb; and lane 8, 0.48 kb); a computer search did not reveal sequences of high homology to the probe. Size markers: (lane 1) Hpa II and Hae III digests of ϕ X174; (lane 3) Alu I digest of pBR322; (lane 7) ³²P-labeled Alu I digest of pBR322.

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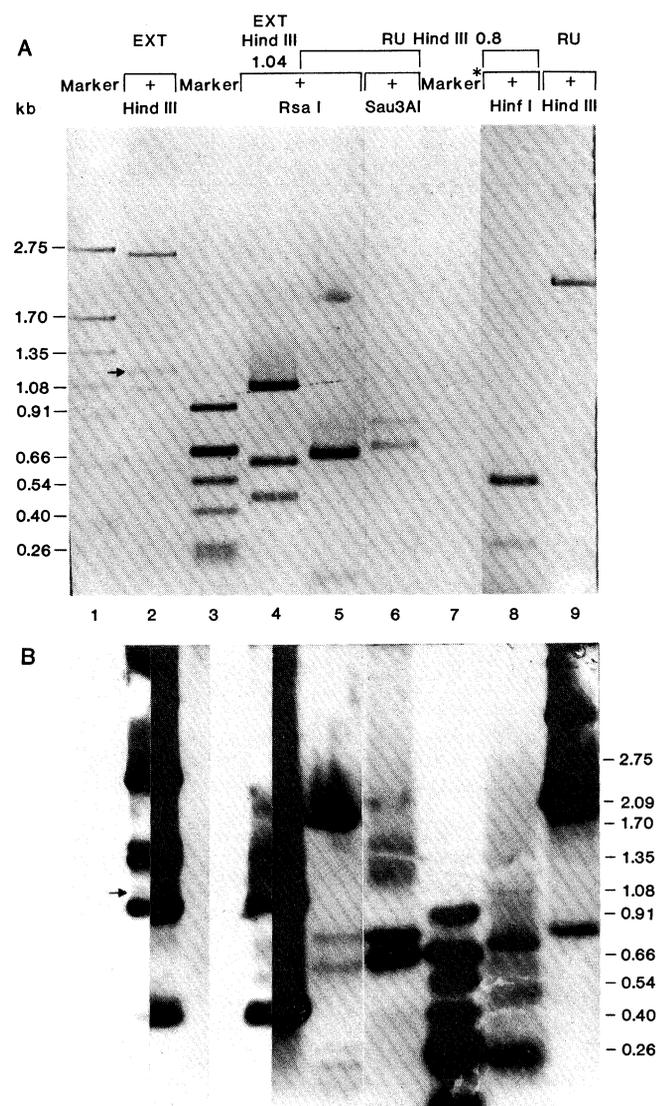


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rounds the amplified sequence of EXT, there are several other repeats (Fig. 3) in this domain of all three variants of the satellite. No major changes occur near these repeated sequences.

Although bovine satellites 1.711a and 1.711b exhibit high homology to bovine satellites 1.706 and 1.715, respectively (4), their repeat units are larger. In both cases, the extra DNA is unrelated to the rest of the repeat unit and is described as inserted (4). In contrast, we describe an expanded satellite repeat unit that results from the amplification of an internal sequence. An inverted tetranucleotide repeat forms the boundaries of the amplified sequence in EXT, the only variant of the three studied to date that contains the amplification. At the site of amplification, the upstream arm of the inverted repeat is missing from the two other variants, RU and TRU.

Other sites of major sequence divergences in this crab satellite DNA also occur in association with domains of repeated DNA's (5, 6). Some of these are homopolymer tracts; others are homopolymers and their variants. Still others contain long tracts of alternating purines and pyrimidines. All can be envisaged as having marked influences on secondary or tertiary structures, or both.

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16. We thank M. Spann for technical assistance and D. Million for computer advice. This work was supported by seed money from Oak Ridge National Laboratory, by grant PCM 78-23373 from the National Science Foundation (to D.M.S.) and by the Office of Health and Environmental Research, U.S. Department of Energy under contract W-7405-eng-26 with Union Carbide Corporation.

19 April 1983; revised 24 June 1983

26 AUGUST 1983

Human Macrophages Armed with Murine Immunoglobulin G2a Antibodies to Tumors Destroy Human Cancer Cells

Abstract. *Macrophages isolated from tumor-bearing patients as well as cultured human monocytes express Fc receptors that cross-react strongly with murine immunoglobulins of the G2a but only slightly or not at all with the G1, G2b, or G3 subclasses. Such macrophages in the presence of murine immunoglobulin G2a monoclonal antibodies to tumors mediated the killing of tumor cells in vitro. These data suggest that monoclonal antibodies of the G2a subclass may be useful in the immunotherapy of human cancer.*

Murine monoclonal antibodies have been used successfully for the detection of cancer antigens in the blood of patients (1-3) or on the surface of tumor cells (4, 5). Such antibodies conjugated with radioactive labels have also been used to locate tumors (by imaging) in patients (6). Specific destruction of human tumor xenografts in nude mice (7, 8) was observed after inoculation of monoclonal antibodies of immunoglobulin G2a (IgG2a) isotypes only (9, 10), although all other isotypes were tested. We have also shown that tumor cells are destroyed as a result of the interaction of IgG2a monoclonal antibodies and mouse macro-

phages (9). Because tumor-specific monoclonal antibodies derived from humans are not yet available for immunotherapy we studied the interaction of murine immunoglobulins with human effector cells in vitro. We examined two basic phenomena: (i) whether human monocytes and macrophages express Fc receptors that cross-react with the various murine IgG subclass proteins and (ii) whether human monocytes and macrophages in the presence of murine monoclonal antibodies mediate lysis of human tumor cells.

We first established that human peripheral blood leukocytes would prevent

Table 1. Expression of Fc receptors for mouse immunoglobulins on human monocytes and macrophages. Human peripheral blood leukocytes (PBL) were prepared by Ficoll-Hypaque centrifugation. The PBL layer was washed twice in phosphate-buffered saline (PBS) containing 20 mM Hepes. The mononuclear cells were counted and standardized to appropriate concentrations in RPMI 1640 medium containing 20 mM Hepes and 0.1 percent autologous plasma or, alternatively, 10 percent fetal calf serum. Portions (1 ml) of PBL were distributed into 24-well tissue culture plates and allowed to adhere for 2 hours at 37°C in a 5 percent CO₂ atmosphere. The nonadherent cells were removed and the adherent monocytes were washed twice in RPMI 1640 medium. More than 97 percent of the adherent cells were monocytes, as determined by Wright's staining, nonspecific esterase activity, and latex phagocytosis. Macrophages from cancer patients were isolated from ascitic or pleural effusions after isolation of all cells by centrifugation. The cells were counted and the percentage of macrophages established by Wright's staining. Cells were standardized to appropriate concentrations in RPMI 1640 medium. Portions (1 ml) were distributed into 24-well plates and after 2 hours processed as above for selection of adherent cells. More than 90 percent of adherent cells were macrophages by the above criteria. Fc receptors were detected by incubating monocytes and macrophages with sheep or ox erythrocytes coated with mouse monoclonal antibodies to sheep red blood cells (established by W. C. Raschke, La Jolla Cancer Research Foundation, and received through ATCC) or to ox red blood cells (established by us). Red blood cells were coated with equivalent amounts of antibodies to saturate binding sites. More than five erythrocytes per cell were considered positive.

Source of human monocytes and macrophages	Donor	Days in culture	Percentage of cells expressing Fc receptors reacting with mouse immunoglobulins				
			G1	G2a	G2b	G3	M
Blood	J.K.	0 to 9	0	0	0	0	0
		10 to 14	0	90	0	0	0
		17 to 40	0	50	0	0	0
	M.K.	0 to 13	0	0	0	0	0
		14	0	20	0	0	0
		18	0	60	< 1	50	0
C.R.	34	0	90	0	0	0	
	0 to 20	0	0	0	0	0	
	21	0	50	0	0	0	
	29	0	90	0	0	0	
Melanoma	WM369*	1	0	50	0	0	0
	WM373†	1	0	50	0	0	0
Colon carcinoma	J.R.†	1	0	55	0	0	0
		3	0	70	0	0	0
		7	0	90	< 5	0	0

*Isolated from pleural effusion.

†Isolated from ascitic effusion.