ture (Fig. 1d) was pumped from the outside lagoon adjoining the lobster hatchery. The animals were fed daily in excess of demand, and uneaten food was removed after 24 hours.

In Fig. 1a the increase in size per molt of lobsters reared at the hatchery at ambient temperature is compared with values predicted by Wilder's equations for growth in Canadian waters (*3*). Our experimental lobsters, reared at elevated temperatures, exhibited increments per molt similar to those predicted by Wilder for wild lobsters.

The significance of the apparent differences for molts 18 and 19 between empirical and theoretical results is unknown. It may be more apparent than real, because of the small numbers of animals studied.

The relation of carapace length (*5*) to weight is shown in Fig. 1b. The increase in weight per molt in these studies can be determined by comparing Fig. 1, a and b.

In addition to his studies of lobster growth in the sea, Templeman (*2*) also measured growth rates of lobsters from stages 4 to 12 at temperatures between 19° and 22°C. The frequency of molting increased at higher temperatures in these limited studies. Hughes and Matthiessen reported later that optimum growth for larval lobsters in stages 1 to 4 is achieved between 20° and 23°C (*6*).

The size increments we observed for the first 12 molts coincided with Templeman's observations for the same growth stage. We transferred 12 lobsters to warm water tanks (20° to 24°C) and observed them for size increment and molting frequency. Rates of weight increase in both warm and ambient seawater at the lobster hatchery (Fig. 1c) are compared with a theoretical curve for growth rate in the sea in the vicinity of Prince Edward Island, Canada. Lobsters reared in warm water grew faster as a result of a shorter intermolt period. Lobsters molt more frequently at elevated temperatures but have the same size increment per molt as do wild lobsters. The apparent difference between Wilder's calculations and our observations of growth rate or molting frequency can be explained by examining the monthly temperatures in the three situations (Fig. 1d). If growth rate is temperature-dependent, it is not surprising that lobsters exposed to the warmer average temperatures on Martha's Vineyard grow to sexual maturity in 5½ years whereas animals growing in the colder Canadian environment take approximately 8 years (*3*).

In nature, most lobsters with carapace lengths of 90 mm have reached sexual maturity. All of the experimental lobsters that have 83-mm carapaces and were raised in warm water to this size have demonstrated normal sexual responses and have mated. Also, the developmental period for fertilized eggs is decreased by 33 percent in warm water.

Encouraged with these results and the potential to mate chosen parents, we tried to select lobsters that grew faster than average or were molting more frequently. More than 100,000 lobster larvae were screened for rapid growth. In two steps we selected 200 and finally 13 individual animals displaying at least a 1.5-fold increase in molting frequency. We have also selected lobsters that are much larger than average for their age. The results of these selections suggest that it may be possible to produce lobsters of marketable size in about 18 months.

It is possible to estimate food conversion for lobsters from these studies. The amount of food offered daily was proportional to body size. When the water temperature dropped below 5°C, control animals ate very little. Conversion factors of 4 : 1 and 11 : 1 were calculated for the animals grown in warm water and ambient seawater, respectively. Uneaten food was not considered in these calculations; therefore, these values are considered to be maximum and tentative conversions. Precise experiments to measure actual food conversion with a variety of inexpensive or synthetic diets are needed. The results of these studies indicate that there may be a lobster farm in the future.

JOHN T. HUGHES
JOHN J. SULLIVAN
Massachusetts Department of Natural Resources, Division of Marine Fisheries, Lobster Hatchery and Research Station, Vineyard Haven 02568

ROBERT SHLESER
Institute for Marine Resources, University of California, Davis 95616

References and Notes

1. P. B. Hadley, *U.S. Fish Wildl. Serv. Fish. Bull.* (1906), p. 153.
2. W. Templeman, *J. Biol. Bd. Can.* **1**, 213 (1936).
3. D. G. Wilder, *J. Fish. Res. Bd. Can.* **10**, 371 (1953).
4. Martha's Vineyard water is taken from a shallow bay, which is subject to seasonal temperature fluctuations that are greater than those normally experienced by natural lobster populations in open coastal waters.
5. Carapace length is the distance from the rear of the eye socket to the rear end of the body shell, measured along a line parallel to the center of the body shell. In Massachusetts a legal-size lobster has a carapace length of at least 81 mm (3 3/16 inches).
6. J. T. Hughes and G. C. Matthiessen, *Limnol. Oceanogr.* **7**, 414 (1962).

2 June 1972

DNA Polymerases from RNA Tumor Viruses and Human Cells: Inhibition by Polyuridylic Acid

Abstract. *Polyuridylic acid inhibited DNA polymerases purified from three species of oncornaviruses as well as three out of seven DNA polymerases purified from cells. Viral and cellular DNA polymerases could not be distinguished by polyuridylic acid inhibition, but were easily distinguished by their template preferences in the presence of magnesium.*

A report appeared suggesting that inhibition by polyuridylic acid [poly(U)] might be useful in distinguishing DNA polymerase (reverse transcriptase) of RNA tumor viruses from cellular DNA polymerases (*1*). That report demonstrated that two cellular DNA polymerases were not inhibited by poly(U), a potent inhibitor of a crude preparation of the RNA tumor viral enyzme (*1*). The possibility that poly(U) might be a specific inhibitor of the oncornaviral DNA polymerase has interesting ramifications, especially for identification of this or a similar enzyme in cells, particularly human neoplastic cells. Tuominen and Kenney (*1*) discussed the possibility of using poly(U) in the detection of oncornaviral DNA polymerases in cellular systems, as well as separating these viral and cellular polymerases by specific elution from DNA-cellulose columns with buffers containing poly(U). Therefore, it was imperative to confirm and extend their observations, particularly those relating to the specificity of poly(U).

We have examined the inhibitory effects of poly(U) on ten partially purified DNA polymerases, three from RNA tumor viruses and seven from cells. Three types of templates were

used: (i) a synthetic hybrid of polythymidylic acid and polyadenylic acid units [poly(dT) • poly(A)], (ii) a synthetic copolymer of alternating deoxyadenylic acid and thymidylic acid units (poly[d(A-T)]), and (iii) salmon sperm DNA which had been subjected to a mild deoxynuclease treatment (activated DNA) (*2*).

The three viral DNA polymerases used in this study were isolated from avian myeloblastosis virus (AMV) (*3*), Rauscher murine leukemia virus (RLV) and Mason-Pfizer monkey tumor virus (M-PMTV) (*4*). These DNA polymerases exhibited template preferences reported for reverse transcriptase (*5*). Seven cellular DNA polymerases were used in this study; one from *Escherichia coli* (*6*), two from human lymphoblasts of patients with acute lymphoblastic leukemia, two from normal human lymphocytes stimulated for 72 hours with phytohemagglutinin (*7, 7a*), and two from NC-37 cells (*8*) infected with M-PMTV.

The DNA polymerases isolated from the three types of human cells are similar to the cellular DNA polymerases described independently by Weissbach *et al.*, Baril *et al.*, and Chang and Bollum (*9*). The two leukemic DNA-dependent DNA polymerases discussed here should not be confused with a third DNA polymerase recently isolated from the same cells. This third DNA polymerase is distinguished from the major normal cellular DNA polymerases 1 and 2 by its ability to transcribe viral 70*S* RNA (*10*).

The effects of poly(U) on the cellular and viral DNA polymerases with poly[d(A-T)] as template are shown in Fig. 1. When conditions similar to those previously reported were used (*1*), poly(U) did not display a specificity for the viral DNA polymerase. As seen in Fig. 1A, each viral DNA polymerase was inhibited by poly(U). However, three of the seven cellular polymerases were also inhibited, as

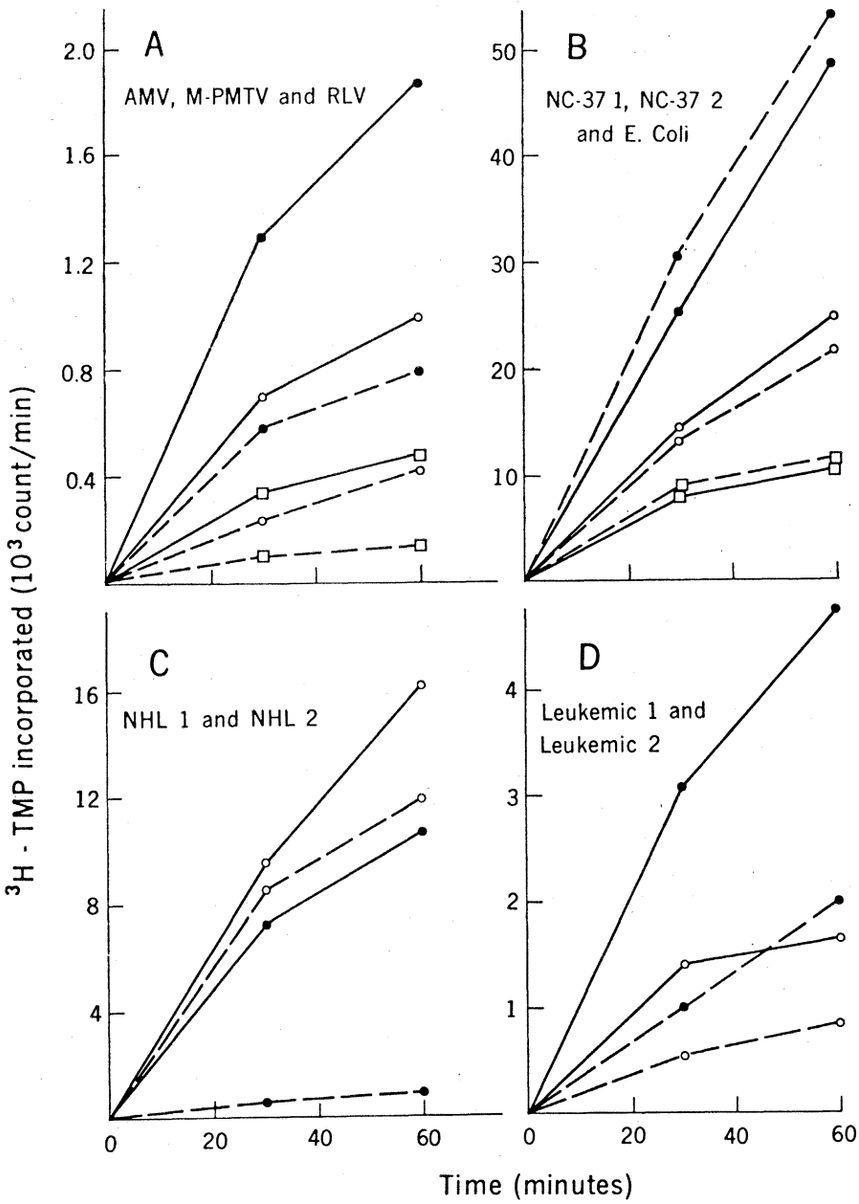

Fig. 1. The effect of poly(U) on DNA polymerases. Viral DNA polymerases were prepared from virus lysed at 0°C with 0.25 percent Triton X-100 in a tris·HCl buffer, *p*H 7.9, containing 0.5*M* KCl. The mixture was sonicated and then centrifuged at 30,000*g* for ½ hour. The supernatant containing the enzymatic activity was subjected to chromatography first on DEAE-cellulose and then on phosphocellulose. In some cases the enzyme that eluted from phosphocellulose was chromatographed on Sephadex G-200 gel (*11*). A minimum purification for the viral DNA polymerases was estimated to be 30- to 50-fold, as judged by activity with the synthetic DNA · RNA duplex, oligomeric thymidylic acid and polyadenylic acid, and minimum detectable amounts of protein (*12*). An estimate of protein concentration was also made by staining electrophoretic gels with Coomassie blue dye (*13*). All human cellular DNA polymerases were purified by a similar procedure. The two DNA polymerases from normal human lymphocytes were designated normal human lymphocyte DNA polymerases 1 and 2, with reference to their order of elution from phosphocellulose (*7a*). The two DNA polymerases from leukemic cells, leukemic DNA polymerases 1 and 2 (*14*), and those from NC-37 cells, NC-37 DNA polymerases 1 and 2, were similarly designated. The two DNA polymerases from normal human lymphocytes were subjected to chromatography on Sephadex G-200 gel after elution from phosphocellulose. Normal human lymphocyte DNA polymerases 1 and 2 were purified 220-fold and 290-fold, respectively (*7a*). Protein concentrations of leukemic DNA polymerases 1 and 2 and NC-37 DNA polymerases 1 and 2 were not measured. The DNA polymerase activity was measured as in Table 1 except that only the poly[d(AT)] template was used (60 μg/ml), and that the concentration of poly(U) was 100 μg/ml. The solid lines represent control activity [no poly(U)], while the broken lines represent activity in the presence of poly(U). (A) AMV DNA polymerase (●); M-PMTV DNA polymerase (○); and RVL DNA polymerase (□). (B) NC-37 polymerase 1 (●); NC-37 DNA polymerase 2 (○); and *E. coli* DNA polymerase 1 (□). (C) Normal human lymphocyte DNA polymerase 1 (●) and normal human lymphocyte DNA polymerase 2 (○). (D) Leukemic human lymphocyte DNA polymerase 1 (●) and leukemic human lymphocyte DNA polymerase 2 (○).

shown in Fig. 1, C and D. Normal human lymphocyte DNA polymerase 1 activity was reduced by 90 to 95 percent; leukemic DNA polymerases 1 and 2 activities were reduced approximately 50 percent. The inhibition of these three cellular DNA polymerase activities is comparable to that obtained for the purified viral DNA polymerases under identical assay conditions. In fact, the greatest inhibition was obtained with the cellular enzyme normal human lymphocyte DNA polymerase 1. Poly(U) had little or no effect on the other four cellular DNA polymerases. *Escherichia coli* DNA polymerase 1 was unaffected, confirming the previous results (*1*). Likewise, poly(U) did not inhibit DNA polymerase 1 from NC-37 cells and had only slight effects on DNA polymerase 2 from both NC-37 cells and normal human lymphocytes.

The relative inhibition by poly(U) with different templates is compared in Table 1. Most of the enzymes were tested at two concentrations of poly(U) with each of three templates, poly[d(A-T)], poly(dT)·poly(A), and activated salmon sperm DNA. The viral polymerase was inhibited to some degree by poly(U) at both concentrations tested with all three templates. When a DNA polymerase was sensitive to poly(U) with the poly[d(A-T)] template, a similar sensitivity was observed with the activated salmon sperm DNA template. Likewise, those DNA polymerases not inhibited by poly(U) with poly[d(A-T)] template exhibited no inhibition with activated salmon sperm DNA template.

In contrast to the results with DNA duplexes as the template, potent inhibition by poly(U) was noted for all ten partially purified DNA polymerases when the template was poly(dT)·poly(A). These data demonstrate that the degree of inhibition by poly(U) may be influenced by certain templates. However, this inhibition might be an effect of poly(U) upon the hybrid template rather than upon the enzyme.

The data in Table 1 imply that the dissociation constant (K_i) of poly(U) for normal human lymphocyte DNA polymerase 1 is lower than that for the tumor virus DNA polymerases tested. Competitive inhibition kinetics between poly(U) and the poly[d(A-T)] template were observed for both the M-PMTV DNA polymerase and normal human lymphocyte DNA polymerase 1. The apparent K_i's for poly(U) with these two enzymes were determined. Expressed in micrograms per milliliter, the K_i with normal human lymphocyte DNA polymerase 1 was 6.2, while that with the M-PMTV DNA polymerase was 20. The difference in these K_i's demonstrates that at least one cellular DNA polymerase has a greater affinity for poly(U) than one oncornavirus DNA polymerase.

Table 1. Inhibition by poly(U) of DNA polymerases with double-stranded DNA or an RNA·DNA hybrid as template. The DNA polymerase activity was measured at 37°C in 50-μl reaction mixtures containing 50 mM tris·HCl, pH 7.6; 5 mM $MgCl_2$; 50 mM NaCl; 5 mM dithiothreitol; 80 μM deoxyadenosine triphosphate, deoxycytidine triphosphate, and deoxyguanosine triphosphate; 5.6 μM ^{3}H-labeled thymidine triphosphate (18,000 count min^{-1} pmole^{-1}) and the DNA double-stranded templates, poly[d(A-T)] and activated DNA at 60 μg/ml and poly(dT)·poly(A) at 20 μg/ml. Poly(U) was present at two concentrations. Reactions were linear up to 30 minutes. Incorporation of [^{3}H]thymidine monophosphate was calculated from 20-minute time points. Generally 10 μl of the enzyme preparation was used in the assay. The reactions were terminated with the addition of 50 μg of carrier transfer RNA and subsequent precipitation with 2 ml of 10 percent trichloroacetic acid containing 0.02M pyrophosphate. The product was collected on Millipore filters and counted in a liquid scintillation counter. All enzymes except RLV(DEAE) were purified through the phosphocellulose chromatography step; RLV(DEAE) denotes the DNA polymerase from RLV which was purified through the diethylaminoethyl-cellulose chromatography step and required added template for activity.

DNA polymerase*	Template	Percent inhibition by poly(U) at 20 μg/ml	Percent inhibition by poly(U) at 100 μg/ml
NHL 1	$d(A\text{-}T)_n$	80	90
	DNA†	75	90
	$(dT)_n\cdot(A)_n$‡	100§	100
NHL 2	$d(A\text{-}T)_n$	0	25
	DNA	10	15
	$(dT)_n\cdot(A)_n$	95§	100
Leukemic 1	$d(A\text{-}T)_n$		55
	DNA		
	$(dT)_n\cdot(A)_n$		100
Leukemic 2	$d(A\text{-}T)_n$		50
	DNA		
	$(dT)_n\cdot(A)_n$		100
NC-37 1	$d(A\text{-}T)_n$	0	0
	DNA	0	0
	$(dT)_n\cdot(A)_n$	90§	95
NC-37 2	$d(A\text{-}T)_n$	0	20
	DNA	0	20
	$(dT)_n\cdot(A)_n$	95§	100
E. coli 1	$d(A\text{-}T)_n$	0	0
	DNA	0	0
	$(dT)_n\cdot(A)_n$	75§	100
AMV	$d(A\text{-}T)_n$	40	60
	DNA		70
	$(dT)_n\cdot(A)_n$	75§	100
M-PMTV	$d(A\text{-}T)_n$	45	55
	DNA	45	60
	$(dT)_n\cdot(A)_n$	60§	100
RLV	$d(A\text{-}T)_n$	50	70‖
	DNA	45	60
	$(dT)_n\cdot(A)_n$	90§	100
RLV(DEAE)	$d(A\text{-}T)_n$	60	90‖

* Designations of the cellular and viral polymerases are given in the text except NHL 1 and NHL 2 which are normal human lymphocyte DNA polymerases 1 and 2. † DNA-native salmon sperm DNA which has been activated by a mild deoxynuclease treatment (*2*). ‡ Poly(dT)·poly(A). § The poly(U) concentration for these determinations was 10 μl/ml. ‖ In agreement with the earlier report (*1*), this crude oncornavirus DNA polymerase was strongly inhibited by poly(U). However, after further purification of the enzyme by phosphocellulose chromatography, inhibition was less.

Differences in sensitivity to inhibition by poly(U) (when the DNA duplex templates were used) might be attributed to ribonuclease contamination of the various DNA polymerases. Ribonuclease in the DNA polymerase preparations could result in either loss or enhancement of inhibition, depending on the properties of the degradation products of poly(U). Three DNA polymerases—normal human lymphocyte 1, NC-37 1, and M-PMTV, were used to test the possibility of a ribonuclease effect. (i) To check for "inactivation" of poly(U), the homopolymer was first treated with NC-37 DNA polymerase 1 before incubation with the M-PMTV polymerase 1 and normal human lymphocyte polymerase 1. The poly(U) thus treated was as inhibitory as untreated poly(U) to both M-PMTV polymerase and normal human lymphocyte polymerase 1, demonstrating that NC-37 DNA polymerase 1 did not contain an inactivator of poly(U), such as ribonuclease. (ii) To eliminate enhancement of inhibition by degradation products of poly(U) as an explanation of the results, we included uridylic acid in the DNA polymerase reaction mixtures at a concentration of 100 μg/ml [the highest concentration of poly(U) tested]. Uridylic acid was not inhibitory for any DNA polymerase tested.

Our findings confirm the inhibitory effect of poly(U) on DNA polymerases of RNA tumor viruses (*1*) and extend this observation to three classes of virus: avian, murine, and primate. However, inhibition by poly(U) is not a specific property of these DNA polymerases since some cellular DNA polymerases are inhibited at least as much as the viral enzymes. Thus, sensitivity to poly(U) cannot be used to distinguish reverse transcriptase from all cellular DNA polymerases.

JOHN W. ABRELL
Bionetics Research Laboratories, Bethesda, Maryland 20014
R. GRAHAM SMITH
MARJORIE S. ROBERT
ROBERT C. GALLO
Laboratory of Tumor Cell Biology, National Cancer Institute, Bethesda

References and Notes

1. F. W. Tuominen and F. T. Kenney, *Proc. Nat. Acad. Sci. U.S.A.* **68**, 2198 (1971).
2. A. Schlabach, B. Fridlender, A. Bolden, A. Weissbach, *Biochem. Biophys. Res. Commun.* **44**, 879 (1971).
3. Dr. J. Beard of Duke University, Durham, North Carolina, supplied the infected chicken plasma from which the AMV was purified by centrifuging on 100 percent glycerol, and then on a preformed continuous sucrose gradient (20 to 60 percent).
4. RLV was obtained from Electronucleonics, Bethesda, Maryland, in 0.01*M* tris, 0.1*M* NaCl, 0.001*M* EDTA, *p*H 7. M-PMTV came from the John L. Smith Memorial for Cancer Research, Pfizer Inc., Maywood, New Jersey, as a pellet. These viruses were either freed of cellular debris by centrifuging in a sucrose gradient or collected as a pellet after centrifugation at 100,000*g*. In either case the subsequent isolation of the viral DNA polymerases did not require modification to yield enzymes of comparable purity.
5. N. C. Goodman and S. Spiegelman, *Proc. Nat. Acad. Sci. U.S.A.* **68**, 2203 (1971); R. C. Gallo, *Nature* **234**, 194 (1971); M. Robert, R. G. Smith, R. C. Gallo, P. S. Sarin, J. W. Abrell, *Science* **176**, 798 (1972).
6. *Escherichia coli* DNA polymerase 1, a gift from Dr. Lawrence Loeb, Institute for Cancer Research, Fox Chase, Philadelphia, Pennsylvania, was prepared as described by T. M. Jovin, P. T. Englund, L. L. Bertsch [*J. Biol. Chem.* **224**, 2996 (1969)].
7. R. C. Gallo and J. Whang-Peng, in *Biological Effects of Polynucleotides*, R. F. Beers and W. Baun, Eds. (Springer-Verlag, New York, 1971), p. 303.

7a. R. G. Smith and R. C. Gallo, *Proc. Nat. Acad. Sci. U.S.A.*, in press.

8. NC-37 cells, obtained from the John L. Smith Memorial for Cancer Research, Pfizer Inc., Maywood, New Jersey, are a lymphoblastoid line initially derived from the blood of a normal human donor [E. M. Jensen, I. Zelljadt, H. C. Chopra, M. M. Mason, *Cancer Res.* **30**, 2388 (1970); H. C. Chopra, I. Zelljadt, E. M. Jensen, M. M. Mason, N. J. Woodside, *J. Nat. Cancer Inst.* **46**, 127 (1971)].
9. A. Weissbach, A. Schlabach, B. Fridlender, A. Bolden, *Nature* **231**, 167 (1971); E. F. Baril, O. E. Brown, M. D. Jenkins, J. Laszlo, *Biochemistry* **10**, 1981 (1971); L. M. S. Chang and F. J. Bollum, *J. Biol. Chem.* **246**, 5835 (1971).
10. R. C. Gallo, P. S. Sarin, M. G. Sarngadharan, M. S. Reitz, R. G. Smith, S. N. Bobrow, in *Molecular Studies in Viral Neoplasia* (Williams & Wilkins, Baltimore, in press); M. G. Sarngadharan, P. S. Sarin, M. S. Reitz, R. C. Gallo, *Nature New Biol.*, in press.
11. J. W. Abrell and R. C. Gallo, in preparation.
12. O. H. Lowry, N. J. Rosebrough, A. L. Farr, R. J. Randall, *J. Biol. Chem.* **193**, 265 (1951).
13. A. Chrambach, R. A. Reisfeld, M. Wyckoff, J. Zaccari, *Anal. Biochem.* **20**, 150 (1967).
14. S. S. Yang, F. Herrera, R. G. Smith, M. Reitz, R. C. Ting, R. C. Gallo, *J. Nat. Cancer Inst.* **48**, 1185 (1972).

15. Supported in part by the Leukemia Society of America, Inc. M.S.R. is a fellow of the Leukemia Society of America, Inc. We thank Miss Lynda Chiodetti for technical assistance.

25 May 1972; revised 10 July 1972 ■

Antigen Solubilized from Human Leukemia: Lymphocyte Stimulation

Abstract. *Soluble antigen was extracted with hypertonic (3 molar) potassium chloride from the malignant cells of seven patients with acute leukemia. The antigen and leukemia cells were used to stimulate autologous patients' and allogeneic normal donors' lymphocytes in mixed lymphocyte cultures. The lymphocytes of six patients showed significant blastogenic responses to autologous antigen. In contrast, the lymphocytes of only one of seven normal donors responded to the soluble antigens. Both patients' and normal subjects' lymphocytes responded to the intact leukemia cells. The use of these antigens should facilitate the study of specific tumor immunity in human leukemia.*

The existence of tumor-associated antigens on human leukemic cells has been suggested by the reports of positive lymphocyte responses to autologous leukemia cells in mixed lymphocyte cultures (*1*, *2*). A point of debate is whether a positive blastogenic response to autologous leukemia cells represents a primary response in vitro or whether it actually represents evidence of preexisting immunity (*1*). As a result of the reported successful extraction of tumor-associated antigens from guinea pig hepatoma cells with hypertonic KCl (*3*), we have applied similar techniques to the extraction of soluble antigens from human leukemia cells. These soluble preparations have been assayed in lymphocyte cultures with both autologous leukemic patients' and allogeneic normal donors' lymphocytes. In addition to demonstrating the feasibility of salt extraction of antigens from human leukemic cells, our data suggest that the in vitro lymphocyte responsiveness represents specific tumor immunity.

Blast cells were collected from the peripheral bloods of seven leukemia patients on admission to the hospital. The cells were collected with the IBM or the Aminco blood cell separators (*4*). The red blood cells were removed by exposure of the collected cells to five volumes of tris-buffered ammonium chloride (*5*). The leukemia cells were suspended in media with 10 percent fetal bovine serum and 10 percent dimethyl sulfoxide and frozen and stored in liquid nitrogen.

For extraction of tumor antigen, the method described by Meltzer and coworkers (*3*) was used with some modifications. Depending on the availability of cells, 3×10^8 to 3×10^{10} leukemia cells were used. Ten milliliters of 3*M* KCl in potassium phosphate–buffered saline at *p*H 7.2 were added to every 3×10^8 cells in screw-cap glass tubes 25 by 200 mm. This mixture was equilibrated at 4°C for 16 hours. The contents were then centrifuged at 100,000*g* for 60 minutes at 4°C. The supernatant was dialyzed against 20 volumes of potassium phosphate–buffered saline for 24 hours, with a change of solution every 8 hours. The dialyzed extract was centrifuged at 40,000*g* for 15 minutes at 4°C. The supernatant was concentrated by ultrafiltration at 4°C, and the concentrate was mixed with an equal volume of 3.8*M* ammonium sulfate for 1 hour at 4°C to precipitate the protein. This mixture was centrifuged at 40,000*g* for 15 minutes at 4°C. The precipitate was dissolved in 5 ml of phosphate-buffered saline, *p*H 7.2, and dialyzed against this buffer for 4 hours. The protein concentration was estimated by measurement of optical density at a wavelength of 280 nm. The final preparation was sterilized by filtration through an 0.45-μm Millipore filter and stored at 4°C.

For the lymphocyte culture studies, peripheral blood lymphocytes were

Table 1. Blastogenic response to autologous leukemia cells (LC) and soluble leukemia cell antigen.

Cell and antigen number	Type of leukemia	Incorporation of ^{3}H* (10^3 count/min)		Stimulation index*		Effect of therapy†
		LC	Antigen	LC	Antigen	
1	AML	1.7	2.4	3.1	5.4	R
2	AML	5.9	21.8	10.8	39.6	R
3	AML	9.2	1.0	30.5	4.4	R
4	AML	19.5	1.6	38.0	6.4	R
5	AML	0	0.62	0	1.1	R
6	ALL	0.72	0.56	3.6	2.8	F
7	CML-BC	0	0.64	0	3.1	F

* Maximum response in autologous or allogeneic serum. † R, remission; F, failure.