Specimen temperatures on release of shock pressure were estimated to be 500° to 1400°C.

Most of the small crystals chosen for examination from the shock-loaded sample transmit light, but single-crystal x-ray photographs show that they are highly mosaic enstatite. Approximately 20 single crystals from samples shocked at a pressure of 1 Mb have been examined by single-crystal x-ray techniques, and none showed the alternating sharp and diffuse spot patterns (*4*) produced by disordered enstatite. A Debye-Scherrer pattern made of a randomly selected sample resembles a mixture of ordered and disordered enstatite; therefore, it is likely that a white powder coating the crystals is disordered enstatite. Some white opaque grains approximately 0.2 mm on edge, which give Debye-Scherrer patterns (Fig. 1) almost identical to those of disordered enstatite from enstatite achondrites, have also been isolated from the sample. Table 1 lists the *d* spacings and intensities of ordered enstatite from Bamle, Norway, disordered enstatite from the Cumberland Falls achondrite, and disordered enstatite produced by a megabar shock event. Disordered enstatite single crystals from enstatite achondrites contain a small amount of twinned clinoenstatite. Thus far we have found no evidence of twinned clinoenstatite in artificially shocked enstatite, but we cannot rule out the possibility that it is present.

Table 1. Intensity data for ordered Bamle enstatite, disordered Cumberland Falls enstatite, and Bamle enstatite shocked at 1 Mb; $CuK\alpha_1$ radiation; wavelength = 1.5405 Å; U, lines from minerals other than enstatite; W, weak lines seen easier on photographs; B, broad lines.

Bamle*		Cumberland Falls*		Shocked Bamle†	
d(Å)	I/I_1	*d*(Å)	I/I_1	*d*(Å)	I/I_1
6.33	< 1	6.3	W	6.4	3
4.43	3	4.41	8	4.44	7
4.028	1		W		
3.314	6	3.30	15	3.29	18
U 3.233	1				
3.175	100	3.17	84	3.18	84
U 3.122	2				
U 3.049	5	2.96			
			28	3.00	33
2.946	16	2.94			
2.878	54	2.87	100	2.88	100
2.832	9	2.82	W		
U 2.746	2				
2.710	10	2.70	W, B		
2.540	25	2.53	26	2.56	20
2.497	18	2.49			
			39	2.48	29
2.477	18	2.47			
U 2.386	< 1				
2.364	1	2.36	W, B		
U 2.320	< 1				
2.283	1	2.28	3		
2.257	3	2.24	W		
2.239	3	2.22	W, B		
2.116	12	2.11			
			21	2.12	20
2.100	12	2.09			
2.060	5	2.05			
			10	2.03	23
2.025	7	2.01			
1.988	9	1.98	7	1.99	20
1.961	11				
1.929	1	1.92	W		
1.888	4				
1.841	4	1.85	W, B		
1.803	1				
1.788	4	1.78	11	1.79	25
1.779	3	1.77	W, B		
1.737	6	1.73	6	1.74	16
1.710	6				
1.681	1	1.67	W, B	1.67	5
1.652	1	1.64	W, B	1.62	18
1.610	11	1.60	14		
1.591	6	1.58	7		
1.529	5	1.53			
			15	1.53	24
1.522	7	1.52			
1.488	23	1.49	12	1.49	10
1.473	17	1.47	21	1.47	30

* Data from Pollack and Ruble (*9*). † Intensities determined from densitometer trace of Fig. 1C.

In our shock pressure and temperature calculations we have assumed that the Hugoniot equation of state for Bamle enstatite corresponds approximately to the Hugoniot equations reported by McQueen *et al.* for Bushveld and Stillwater bronzites (*5*). The pressure estimates are insensitive to large deviations from the assumed Hugoniot equation. The postshock temperature estimates, however, are quite sensitive to both the Hugoniot equation and to the release adiabat, that is, the path followed by the material (in the pressure-volume plane) on release of pressure. The large uncertainty in the estimated postshock specimen temperature is the result of calculations of the limits of possible values and primarily reflects the lack of any experimental data on the release adiabat for pyroxenes.

At present, we are unable to determine whether the disordered enstatite found in the shock-loaded samples resulted from high shock pressures, from high postshock temperatures, or perhaps from the combination of the two. We cannot exclude the possibility that the enstatite samples were at sufficiently high temperature on release of pressure to be in the protoenstatite stability field (*6*) and that the disorder could have been more easily produced by a simple thermal cycle.

Additional evidence indicates that many meteorites, both irons and stones, have experienced shock pressures of about a megabar (*7*). We suggest that most occurrences of disordered orthopyroxene in enstatite and eucrite achondrites can be interpreted as the result of high shock pressures, but we would emphasize that the presence of disordered orthopyroxene in meteorites, especially chondrites (*8*), is not unequivocal evidence of shock damage.

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1. The term "disorder" has been used to describe two different structural aspects of pyroxenes: (i) stacking and (ii) Mg-Fe disorder. Here it refers to stacking disorder.
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24 March 1969; revised 12 May 1969

Macrophage Ribonucleoprotein: Nature of the Antigenic Fragment

Abstract. *The antigenic fragments of bacteriophage T2 recovered in RNA derived from macrophages infected with T2 bacteriophage retain their capacity to combine with specific neutralizing antibody to T2. The preservation of the complete native tertiary structure of tail fiber antigen of bacteriophage T2 is not required for immunogenicity.*

An unusual ribonucleoprotein of macrophages has the ability to combine with antigens to which these cells are exposed. This antigen-ribonucleoprotein complex can induce the production of specific antibody against the challenging antigens (*1*, *2*). Some features of this complex of antigen and ribonucleoprotein (RNP) are a unique density in cesium sulfate solution (1.588 g/cm^3), a protein content of 28 percent, and an approximate molecular weight of 12,000 (*3*). These facts indicate that the maximum size of the antigenic frag-

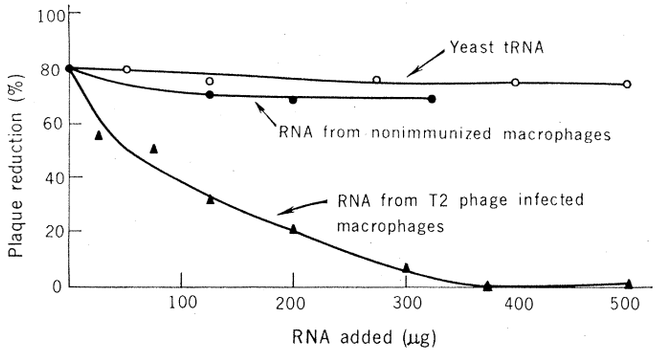

Fig. 1 (left). Adsorption of neutralizing antibody to T2 by macrophage RNA. Adsorption was carried out with variable amounts of RNA from T2-infected macrophages, RNA from noninfected macrophages, or yeast tRNA against a standardized dilution of antiserum to T2. This antiserum, when unadsorbed, inhibited the appearance of 79 percent of a standard input of 289 plaque-forming units of T2 bacteriophage. Percentage of plaque reduction after adsorption of standardized antiserum with variable amounts of RNA from different sources is also shown.

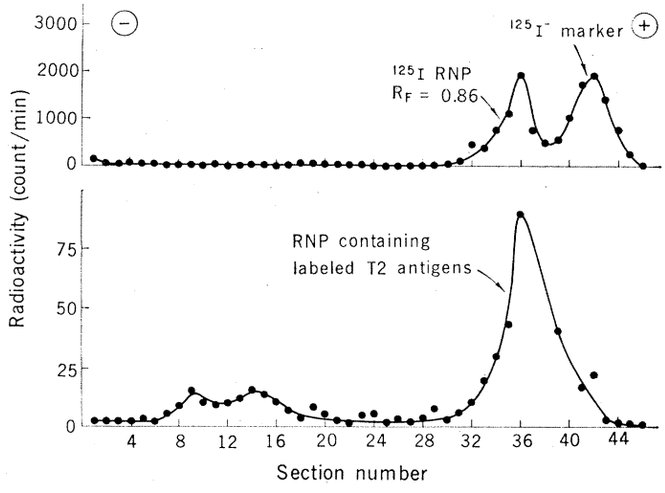

Fig. 2 (right). RNA (150 μg) derived from macrophages infected with ^{125}I-labeled T2 bacteriophage was subjected to polyacrylamide gel electrophoresis in parallel with another gel containing purified RNP labeled in the protein moiety with ^{125}I and a ^{125}I-labeled NaI marker to identify the solvent front.

ment in this complex is 30 to 35 amino acids. Linkage of antigen to RNP is referred to here as "processing" of antigen.

Certain antigens require full preservation of their complete tertiary structural conformation for immunogenicity. Among antigens in this group are lysozyme, insulin, and ribonuclease (*4*). Catabolism of these antigens by macrophages would be expected to destroy the configuration of the antigenic molecule resulting from the interaction of side-groups or helices of adjacent regions of the molecule. Small regions of localized tertiary structure might be preserved if degradation were limited or if certain tertiary antigenic determinants were selectively excised. Nevertheless, those antigens whose immunogenicity depends on preservation of native tertiary structure would be expected to lose their immunogenicity through processing. However, in several well-studied cases (*5, 6*), linkage of antigen to RNA appears to occur, and in these cases, the immunogenic capacity of the antigen is enhanced by being converted to an antigen-RNA complex.

The localization of immunogenicity for the tail fiber of the T2 bacteriophage in the macrophage RNP complex has been studied (*2*). The T2 bacteriophage antigenic residues in this complex are not large enough to contain substantial regions of native tertiary structure. An important prediction from the earlier studies (*2*) is that the antigenic residues derived from T2 bacteriophage should be able to recognize their specific neutralizing antibodies, despite the absence of large-scale tertiary structure. A corollary of this prediction is that the antigenic fragment responsible for producing neutralizing antibody to T2 tail fiber does not depend on extensive preservation of tertiary structure for either immunogenicity or antibody recognition.

To obtain antiserum to T2, white Wistar rats were immunized with three intraperitoneal injections of 10^{10} plaque-forming units (PFU) of bacteriophage T2 and were bled for antiserum 3 weeks after the last dose of antigen. Macrophages were obtained from the abdominal cavities of white Wistar rats (180 to 220 g) by drainage, 5 days after an intraperitoneal injection of Bayol-52 (Esso) white oil. Approximately 85 to 90 percent of the cells were of the large mononuclear type. These cell populations were incubated for 1 hour at 37°C with ^{125}I-labeled bacteriophage T2 (2×10^6 count/min per microgram of T2 protein); the ratio of bacteriophage to macrophage was 20 to 1. After incubation, RNA was prepared from these cells (*2*). The specific activity of the RNA was 29 count min^{-1} μg^{-1}. The distribution of antigen in the RNA population was evaluated by electrophoresis of the labeled RNA on 10 percent polyacrylamide gels; the discontinuous buffer system of Richards *et al.* was used (*7*). The gels were sliced into 1.3-mm fragments after electrophoresis, and radioactivity was assayed (*8*).

For the purpose of determining whether the T2 antigenic fragments contained in this RNA preparation could specifically adsorb antibody to T2, samples (0.1 ml) of RNA (250 μg/ml) were initially incubated with 0.3*M* KOH at 37°C for 20 hours to digest the RNA to mononucleotides and to free the antigenic fragments from the RNA carrier. After neutralization, the sample (adjusted to 0.2 ml) was incubated with antiserum (0.5 ml of a 10^{-4} dilution) to T2 for 2 hours at 37°C. Controls of digested transfer RNA (tRNA) and RNA from macrophages not exposed to T2 bacteriophage were carried along in parallel. After incubation, assay of the reaction mixture for neutralizing antibody to T2 was carried out (*9*). Similar studies were carried out against an antiserum derived from a rat immunized with bacteriophage R17, which does not cross-react with T2 (Table 1).

Alkali-digested RNA from macrophages infected with T2 exhibited the capacity to reduce the neutralizing titer of a standard antiserum, as indicated by the rise in plaque-forming units after incubation of antiserum with this RNA. Minimum reductions in neutralization titer were observed after incubation of antiserum with alkali-digested yeast RNA or alkali-digested RNA from uninfected macrophages. These results indicate that the fragments of T2 bacteriophage antigens present in RNA extracted from T2-infected macrophages represent degradation products of whole T2 proteins which are capable of reacting with neutralizing antibody. Thus, neither the original tertiary

Table 1. Specific adsorption of neutralizing antibody by fragments of bacteriophage T2 antigens in RNA extracted from T2-infected macrophages. One-tenth milliliter of a solution of RNA (250 μg/ml) from T2-infected macrophages was made 0.3*M* in KOH and incubated at 37°C for 20 hours. After neutralization, the sample volume was adjusted to 0.2 ml, and 0.5 ml of a 10^{-4} dilution of a standard antiserum to T2 serum was added. Adsorption was carried out for 2 hours at 37°C. The reaction mixture was then assayed for residual antibody activity by scoring for the ability of the reaction mixture to inactivate a standard number of bacteriophage as described (*10*). An equal amount of this RNA was treated with Pronase prior to alkali digestion and carried through in an identical manner, as were 25-μg samples of RNA from macrophages not exposed to T2 bacteriophage and of yeast RNA. The samples of RNA's were tested in two separate experiments against antiserum to T2 with phage T2 and, in one experiment, against antiserum to R17 with phage R17. Plaque input refers to the standard number of plaque-forming units of each bacteriophage used in the assay.

Fragment treatment	Phage counts		
	T2		
	Experiment a	Experiment b	R17
Plaque number input	285	312	320
Adsorbed with alkali-digested RNA from T2-treated macrophages	141	186	48
Adsorbed with alkali-digested RNA from macrophages not exposed to T2 bacteriophage	84	107	52
Adsorbed with alkali-digested yeast RNA	87	117	59
Adsorbed with Pronase-treated and alkali-digested RNA from T2-infected macrophages	58	121	47
Unadsorbed antiserum	56	116	49

structure of these antigens nor the RNA carrier of the RNP complex is required for recognition of specific neutralizing antibody by the tail fiber antigens of T2 bacteriophage.

If increasing amounts of RNA from T2-infected macrophages were used to adsorb the standardized neutralizing antiserum to T2, all of the total neutralizing antibody could be removed by this RNA (Fig. 1). In contrast to the result with RNA from T2-infected cells, the use of larger amounts of yeast RNA or RNA from noninfected macrophages did not result in significant loss of neutralizing antibody from the standardized antiserum. Thus, all of the neutralizing antibody formed by immunization of rats with intact T2 bacteriophage can be adsorbed by fragments of T2 bacteriophage antigens in the RNA derived from macrophages previously exposed to T2 bacteriophage. The fragments of antigen in this RNA are capable of inducing neutralizing antibody against the intact bacteriophage (*1*). The precise role of the antigenic fragments linked to macrophage RNA in the antibody response in vivo is not clear. The data are compatible with the suggestion that the processing of T2 antigens by macrophages is a pathway (perhaps the principal pathway) by which T2 antigens are presented to the cell destined to form antibody.

Furthermore, the labeled antigenic fragments in RNA derived from macrophages infected with ^{125}I-labeled T2 are distributed in the RNP complex (Fig. 2). Nearly all of the labeled antigen is found to have the same R_F on 10 percent polyacrylamide gels as that of RNP which has been purified by chromatography on tetraethylaminoethyl (TEAE)-cellulose (*2*). This result is similar to that obtained with a soluble immunogenic synthetic copolymer poly L-(Glu^{50}Ala^{40}Tyr^{10}), in which the distribution of labeled antigen in the whole RNA extracted from macrophages exposed to this polymer is exclusively resident in the RNP fraction (*10*). Examination of cesium sulfate density gradients of macrophage RNA derived from macrophages infected with ^{125}I-labeled T2 phage reveals a distribution of small polypeptide fragments along the gradient (*2*). In view of the distribution of antigen shown in Fig. 2, it would appear likely that these fragments are released from the RNP complex in solutions of high ionic strength. Thus, the antigens are not randomly distributed in the RNA population.

The presence in RNP of T2 antigenic fragments which specifically react with neutralizing antibody indicates that these fragments represent small fractions of the whole T2 proteins and that the preservation of extensive tertiary structure of these antigens is not necessary for recognition of specific antibody. These results are in agreement with the finding of complement-fixing T2 antigens in RNA from T2-infected macrophages by Friedman *et al.* (*6*) and with our previous studies which suggested that the T2 antigens present in macrophage RNA represented digested fragments of the original T2 proteins. The data do not rule out the possibility that some of the T2 antigens present in the RNP may not have their antigenic specificity preserved. However, the demonstrated immunogenicity of the T2-RNP (*1*) must be attributable to those antigenic fragments whose specificity is preserved. Whether the processing of T2 bacteriophage antigenic fragments by macrophages is an obligatory step in the formation of antibodies against T2 bacteriophage remains to be shown.

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11. Supported by NIH grant AI 08307 and by a grant from the Medical Foundation of Boston.

5 May 1969

Temperature-Dependence of Resistance at an Electrotonic Synapse

Abstract. *The junctional resistance at septa of the crayfish lateral giant axon is inversely related to temperature with a* Q_{10} *of about 3 over the range from 5° to 20°C. Nonjunctional axonal membrane is much less affected. Resistance changes occur rapidly with temperature changes. No correlates in ultrastructure of the synapses have been found.*

Transmission of impulses across a septum of the lateral giant axon of crayfish (*Procambarus*) is electrically mediated, the septum behaving like a fixed resistance inserted in the path of current along the axonal core (*1*). Each segment of the fiber is a separate axon with its own cell body (*2*); electrotonic synapses connecting successive axons are located in the septa. Morphological studies indicate