solved" (*3*, p. 111). The removal of tritium from the blanket for injection into the plasma seems to be relatively easy. The cost of such a system at 1000 Mw_e at this early date appears to be low; estimated capital costs are \$150/kw_e—equivalent to about 2.6 mill/kw · hour (*1*, p. 27). The cost per kilowatt-hour (electrical) for thermonuclear power may be substantially less than that for: advanced converters, about 4 mill/kw · hour (*5*); breeders, less than 4 mill/kw · hour (*6*). Since a long confinement time of high-temperature plasma has not yet been experimentally demonstrated, these estimates for thermonuclear power are only first approximations and can be expected to change as knowledge of the system grows.

For the fusion reactor, the fission products and induced activity wastes from the alpha particles and neutrons generally will be negligible; the major problem will be loss of tritium. If Rose's estimate is correct (*7*)—that 1.15 tritons (T) must be generated for each triton burned, because of losses and adsorption—10^6 c of tritium must be disposed of daily per 1000 Mw_e of power produced daily.

The conceptual design of Homeyer states that the fusion of 228 g of tritium daily is required for 300-Mw_e production of power (*4*, p. 69):

$$\frac{228\ \text{g T}}{300\ Mw_e \cdot \text{day}} \times 9.78 \times 10^5 \frac{\text{cT}}{\text{gT}} \times 0.15 \text{ loss} \times 1000\ Mw_e = 1.1 \times 10^6\ \text{c T/day}$$

For a fusion reactor, then, tritium would be released at 1.1×10^6 c/day per 1000 Mw of electrical power. This is a significantly higher release of tritium than that to be expected from a fission reactor. The work of Albenesius (*8*) shows that tritium is a fission product, although produced in very low yield (1.15 tritons per 10^4 fissions).

Recent studies (*9*) of the siting of fuel-reprocessing plants indicate that fission-product gases will impose the most severe limitations on siting. One can compare the total amount of tritium produced in fission reactors with that lost from fusion reactors. However, I must emphasize that the major amounts of tritium are released not at the reactor site but during reprocessing of the irradiated fuel elements. In this case, looking at a global average, we should compare the total products per 1000 Mw_e from fission reactors with the losses from a 1000-Mw_e fusion reactor. Only the losses need be considered in a fusion reactor, because the major portion of the tritium is used as fuel. If one assumes the usual factors of 200 Mev per fission, 1.15 tritons per 10^4 fissions, and thermal efficiency of 30 percent for 1000 Mw_e,

$$\frac{1.15\ \text{T}}{10^4\ \text{fissions}} \times \frac{1\ \text{fission}}{200\ \text{Mev}} \times \frac{6.24 \times 10^{18}\ \text{Mev/sec}}{Mw_t} \times \frac{1000\ Mw_e}{0.3\ Mw_e/Mw_t} \times 8.64 \times 10^4\ \text{sec/day} = 1 \times 10^{21}\ \text{T/day}$$

$$\frac{1 \times 10^{21}\ \text{T/day}}{\frac{6.02 \times 10^{23}\ (\text{T/g mol. wt})}{3\ (\text{g/g mol. wt})}} \times 9.78 \times 10^3 \frac{\text{c T}}{\text{g T}} = 50\ \text{c T/day for 1000}\ Mw_e$$

Thus a fission reactor would generate 50 curies of tritium daily per 1000 Mw of electrical power. It appears that the problem of radioactive waste, in terms of production and losses of tritium, will be 2×10^4 times greater with fusion reactors than with fission reactors.

It has already been calculated that the overall increase by the year 2000 in dose rate for the population, due to plants reprocessing nuclear fuel, will be about 2×10^{-3} mrem/year (*10*). If the releases were to be increased by a factor of 2×10^4, the resultant increase in annual dose would not be acceptable; thus methods for removing tritium must be improved in order to make fusion power safe.

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Cell Lysis: Another Function of the Coat Protein of the Bacteriophage f2

Abstract. *Evidence is presented that the coat protein of bacteriophage f2 causes the lysis of infected* Escherichia coli. *To lyse bacteria, the coat protein produced must be of the quality to produce phage particles, although it need not be produced in amounts sufficient to give a large yield of particles. Mutants that are blocked in coat-protein synthesis, or that direct the synthesis of an imperfect coat protein, do not lyse their host bacteria. In addition to its obvious structural role and its postulated regulatory role, another, perhaps enzymatic, role has been found for the coat protein of the phage f2.*

In order to be released from their host bacteria, many bacteriophages cause the production of a lysozyme-like endolysin that dissolves the bacterial cell wall (*1*). However, certain phage mutants, presumably those mutated in the lysozyme gene, result in a normal yield of phage which is not released from the cell and must await artificial rupture (*2*). The small RNA bacteriophages such as f2 also are released by cellular lysis (*3*), but the mechanism of this release is not known. Of the three known genes in f2 (*4*), none is directly related to cell lysis. Gene 1 specifies the viral RNA polymerase; gene 2, a particle assembly protein; and gene 3, the major coat protein. We now have evidence that the coat protein of the phage is responsible for cell lysis.

In an early study of *amber* mutants of bacteriophage f2, when little was known about them, their suppression, or of the genes of f2, Zinder and Cooper (*5*) found that all of their mutants could be classified on the basis of the mode of growth of nonpermissive cells infected with the various mutants. One class of mutants caused the cells to lyse at the normal time, resulting in the release of a full yield of defective phage particles. These mutations are in the assembly gene and allow normal production of coat protein, but produce only defective particles. Another class of mutants allows the infected cells to grow as if uninfected; these are either mutants of the polymerase gene or polar mutants (see below) of the coat-protein gene. After infection with a third class of mutants, the cells increase in mass at a normal rate for a period equivalent to that prior to normal lysis;

Table 1. Properties of *amber* suppressor strains of *Escherichia coli* (*6, 7, 14*).

Bacteria	Amino acid inserted	Suppression (%)
Su⁻		<1
Su-I	Serine	63*; 30†
Su-II	Glutamine	30*; 14†
Su-III	Tyrosine	51*; 55†

*Kaplan *et al.* (*7*). †Garen *et al.* (*14*).

growth then ceases, and lysis does not occur. This third class of infected cells is superficially analogous to those infected with mutants in the "lysis" genes of other phages. However, these cells contain neither viable nor defective phage particles. The phage mutants of the third class are nonpolar mutants (see below) of the coat gene. Thus one might tentatively conclude that lysis occurs only when coat protein is produced (infection with wild-type f2 or with mutants in the assembly gene) and that coat protein is thus required for cellular lysis.

To test this hypothesis further, we made use of new information about *amber* mutants and their suppression (*6, 7*). When not suppressed, *amber* mutations result in premature termination of protein chains. When they are suppressed, depending upon the suppressor strain used, different amino acids are inserted at the site of the *amber* mutation and whole protein is produced in smaller than normal amounts, since suppression is always incomplete (Table 1). We could therefore determine whether insertion of different amino acids at varying efficiencies might alter the assumed relation between coat protein and cell lysis.

The ability of phage to lyse its host bacteria is most conveniently followed by measuring the optical density of infected cultures. Figure 1 shows the results obtained after infection of the different host bacteria with wild-type phage or with the two coat mutants S6 (a polar coat mutant) and S70 (a nonpolar coat mutant). Figure 1A shows that Su⁻ bacteria infected with the polar coat mutant continue to grow almost as well as uninfected bacteria. When infected with the nonpolar coat mutant, they cease growing about 35 minutes after infection, but do not lyse. Parts C and D of the figure indicate that both mutants lyse Su-II (glutamine), but neither lyses Su-III (tyrosine).

To understand these results, we refer to the nature of the two coat mutations and to events occurring after infection of these different strains with the two mutants. Mutant S6 has an *amber* codon at the position in the gene specifying amino acid site 6 from the amino-terminus of the coat protein (*6, 8*); that of S70 is at site 70 (*9*). Coat protein from wild-type phage contains a glutamine residue at each of these positions (*10*). Table 2 summarizes several events after infection of the various suppressor strains with S6 and S70 (*11*). The yield of progeny of the two mutants on the Su⁻ host is of the same low order, but other events following infection are totally different. The failure of S6 to cause the production of

Table 2. Events occurring after infection by mutants S6 or S70 of bacterial strains carrying different *amber* suppressor genes.

Suppressor	Yield* (No. of phage)	Enzyme†	Double-stranded RNA†
	S6 phage		
Su-I	2000	2	2
Su-II	1–10	7	18
Su-III	1–10	8	9
Su⁻	0.01	<0.1	<0.1
	S70 phage		
Su-I	1500	2	5
Su-II	1–10	5	17
Su-III	1–10	18	14
Su⁻	<0.01	10	20

* Progeny phage per infected cell at 60 minutes after infection. † Adapted from Lodish and Zinder (*11*). Refers to the amount of these materials found 40 minutes after infection relative to the amount found after infection with wild-type phage.

enzyme and double-stranded RNA shows that this mutant is polar in vivo. Its polarity has been partially explained by the failure of any of the genes on this RNA message to be translated when it is used as a message in protein-synthesizing extracts of Su⁻ bacteria (*12*).

When Su-I is the host, nearly normal events ensue. However, when Su-II is the host, there is somewhat of a paradox. The amino acid which is inserted by this suppressor strain is glutamine, which should be adequate for the production of phage since it is the amino acid present in the wild-type coat protein at the mutant sites. The explanation for the small yield of progeny phage must lie in the fact that Su-II is the least efficient of the suppressors,

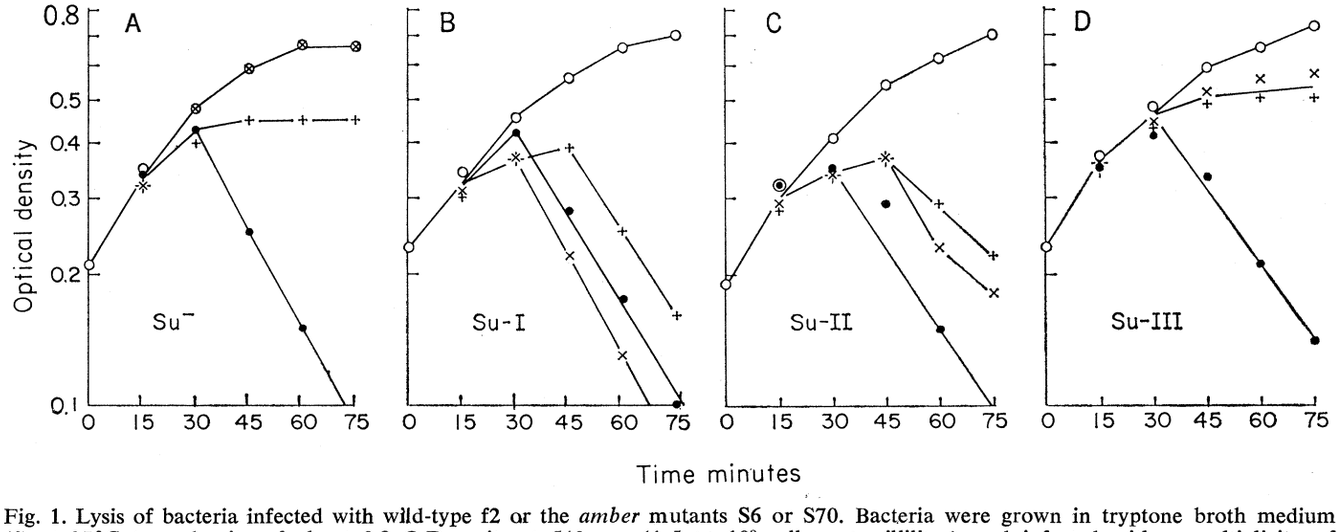

Fig. 1. Lysis of bacteria infected with wild-type f2 or the *amber* mutants S6 or S70. Bacteria were grown in tryptone broth medium (*3*) at 37°C to a density of about 0.2 O.D. units at 540 mμ (1.5×10^8 cells per milliliter) and infected with a multiplicity of five phage per bacterium. Optical density (O.D.) was measured as a function of time. The f2 mutants S6 and S70 are the mutants previously designated Sus-3 and Sus-11, respectively (*5*). The bacterial strains used (*15*) were K140 (Su⁻), K141 (Su-I), K142 (Su-II), K143 (Su-III). Uninfected cells, —○—○—; S6, —×—×—; S70, — + — + —; wild-type f2, —●—●—.

and therefore only a small amount of intact coat protein would be produced. We also note a hyperproduction of polymerase. The presence of this excess enzyme further depresses phage yield by converting newly synthesized single-stranded phage RNA to double-stranded RNA, thereby preventing its entering finished phage particles. From such data Lodish and Zinder (*11*) concluded that the coat protein plays a role as a "repressor" substance in modulating the frequency of translation of the different genes on the phage RNA molecule, a hypothesis for which there is some direct evidence (*13*).

The events occurring after infection of Su-III are not unlike those after infection of Su-II, but the same explanation is not applicable because suppression by Su-III has been at least as efficient as that by Su-I (*6, 7, 14*). We infer that the tyrosine residues inserted by Su-III are incompatible with the normal functioning of the coat protein. The tyrosine-containing proteins can neither form phage particles nor "repress" polymerase production, if these are indeed separate phenomena. Hence the overproduction of polymerase with all of its consequences.

Thus in our experiments the bacteria lysed when the kind of coat protein produced was capable of forming phage particles (S6 or S70 in Su-I) even though the amount of coat protein was lower than normal (S6 and S70 in Su-II), although lysis seems to be somewhat delayed in this latter case (Fig. 1C). When no coat protein is made or when protein incapable of forming phage particles is made (S6 and S70 in Su$^-$ or Su-III), the cells do not lyse. We explain those instances where cell growth ceases (S70 in Su$^-$, S6 and S70 in Su-III) as being a result of the continued synthesis of large amounts of phage RNA, ultimately depleting the cells' own necessary synthetic capacities.

The data presented above support the hypothesis that the coat protein of the RNA phage is required for the lysis of the host bacteria. We do not know whether it performs this lytic function directly or indirectly, nor do we know whether the ultimate target of its action is cell membrane or cell wall. So far, we have not been able to show in vitro any effect of coat protein on a variety of substrates.

Genes are known to specify the structures of proteins which have enzymatic, structural, and regulatory roles in cellular economy. Surprisingly, the single polypeptide chain specified by the "coat protein" gene of the phage f2 appears to have all three kinds of function.

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Sulfate Reduction in Soil: Effects of Redox Potential and pH

Abstract. *Reduction of sulfate to sulfide in waterlogged soils was dependent on soil redox potential and pH. Little or no sulfide accumulated with a redox potential above −150 millivolts, or with a* pH *outside the range 6.5 to 8.5.*

Low oxidation-reduction (redox) potentials are necessary for bacterial reduction of sulfate. As pointed out by Postgate (*1*), the *Desulfovibrio* organisms largely responsible for dissimilatory reduction of sulfate are more exacting in their requirement of anaerobic conditions than are most anaerobes; he noted that a potential in the medium of about −200 mv was required for the initiation of sulfate reduction. Harter and McLean (*2*) found sulfide formation from sulfate in a soil to be very active with a redox potential below −75 mv. Takai and Kamura (*3*) reported that sulfate reduction in waterlogged soil takes place with a redox potential ranging from 0 to −190 mv. In all reported studies of sulfate reduction in waterlogged soils, the redox potential was not controlled during the course of the experiments. In our study, reduction of sulfate to sulfide was examined in two soils under conditions of closely controlled redox potential.

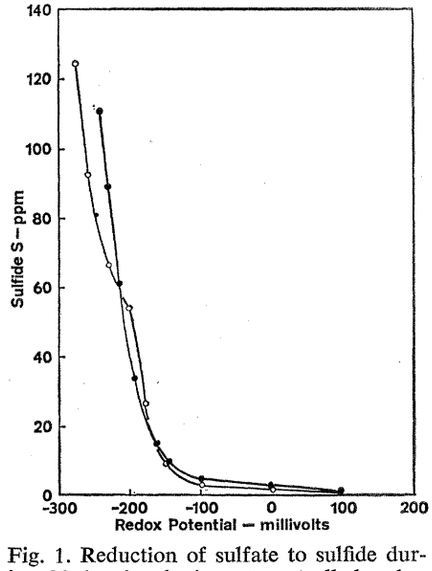

Fig. 1. Reduction of sulfate to sulfide during 30-day incubation at controlled redox potentials in Crowley (●——●) and in Hockley soil (○——○).

The procedure used for control of redox potential in waterlogged soils [reported (*4*)] consists in automatically supplying oxygen to saturated soil cultures when the redox potential falls below the selected value. We used two soils commonly used for rice: a Crowley silt loam from Louisiana and a Hockley very fine sandy loam from Texas. Samples (75-g) of soil containing 0.5 percent of finely chopped rice straw and sulfate sulfur at 100 parts per million (ppm) were incubated for 3 weeks with equal weights of water at controlled redox potentials. After incubation the amount of sulfate that had been reduced to sulfide was determined. The potentials selected ranged from +100 to −250 mv.

The results (Fig. 1) show that redox potential was a controlling factor in the reduction of sulfate to sulfide. In