Heat Sensitivity of *Haemophilus influenzae* Containing Defective Prophage

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Received for publication 19 August 1977

Strains of *Haemophilus influenzae* that carry a defective prophage are more sensitive to heat than is a strain that does not, in the presence of a rec-l mutation, which normally renders prophage noninducible. The prophage of HP1c1, a nondefective phage, does not affect the heat sensitivity.

The commonly used varieties of *Haemophilus influenzae* Rd contain a defective phage (4, 9), which is readily induced by mitomycin C or ultraviolet radiation, except in rec-l recombination-defective mutants (8). This phage, which has no known host, is probably defective in its ability to attach to cells because of its tail plate morphology (9). An ultraviolet-resistant Rd mutant strain, BC200 (1), is also not inducible for defective phage (2, 4), although it is normally inducible after lysogenization with HP1c1 phage (3). It also differs from the wild type in decreased hybridization between its deoxyribonucleic acid and deoxyribonucleic acid extracted from defective phage (4).

A comparison of the heat inactivation of these various strains is shown in Fig. 1. The three strains shown in Fig. 1a were heated together. Strain Rd is more sensitive than is BC200, and the presence of the HP1c1 prophage in BC200 does not affect the sensitivity. Similarly, the three strains shown in Fig. 1b were heated at the same time, and all have the same sensitivity. The presence of neither the HP1c1 prophage nor the rec-l mutation alters the sensitivity.

My interpretation of these observations is that induction of defective phage in the wild-type Rd strain, its HP1c1 lysogen, and the rec-l strain causes these cells to be more sensitive to heat than BC200 or its HP1c1 lysogen. Since rec-I is not inducible by mitomycin C or radiation, but appears to be inducible by heat, it is likely that the repressor for the defective phage is heat sensitive. The repressor of HP1c1, however, is apparently not heat sensitive, since the HP1c1 prophage does not increase the sensitivity of BC200 (Fig. 1a).

Further evidence that there is some induction of defective phage in the heated samples is shown in Fig. 2. BC200 and Rd were placed at 42°C, and the optical density of the cultures was measured at intervals. The Rd curve flattens out at about 80 min after the shift to 42°C and is reminiscent of the similar curve that results when this strain in the competent state has been previously exposed to *Haemophilus parainfluenzae* deoxyribonucleic acid, an effect shown by electron microscopy to be accompanied by the release of defective phage (7). The time at which the break in the curve appears is consistent with the latent period of the defective phage after mitomycin C induction (4). The data of Fig. 2 suggest that only a small fraction of the cells release phage at this time. One possible explanation is that at 42°C not only are phage induced, but also the ability of the cells to produce phage is impaired.

The presumed induction of defective phage in some of the heated cells is somewhat analogous to the induction of the lambda mutant c1857ind (10), except that in the latter case all the killing of the cells at the inducing temperature, 40°C, results from phage production, whereas only part of the killing of Rd can result from induction of defective phage at 42.5°C.

![Fig. 1](http://jb.asm.org/)

**Fig. 1.** Effect of heat on the colony-forming ability of five strains of *H. influenzae*. (a) 42.5°C; (b) 42.0°C.
since there is considerable killing of BC200 without phage induction (Fig. 1a).

The lambda phage mutant is normally inducible by heat in recA mutants, although wild-type lambda prophage is not inducible by irradiation of the recA lysogen (5). This is similar to the apparent heat induction of defective phage in the rec-1 mutant of H. influenzae, which in some but not all respects is similar to recA (6).

This research was carried out at Brookhaven National Laboratory under the auspices of the U.S. Energy Research and Development Administration.

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