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# The Specificity of Lamboid Phage Late Gene Induction (Lamboid Phage Late Gene Specificity)

The purpose of the work reported here was to determine the specificity of late gene induction of phages λ and φ80 by various temperate coliphages. As there is a large and variously related family of coliphages (*1*, *2*), it is of interest to determine whether any of them are able to induce the late genes of their relatives.

These measurements were made possible by the properties of phage φ80*dara* and λ*hy*80*dara*. In both of these phages, genes of the arabinose operon of *Escherichia coli* have replaced some phage late genes and are now controlled as phage late genes (*3*). On φ80*dara* they are under control of the φ80 late gene promoter, and on λ*hy*80*dara* they are under control of the λ late gene promoter (*3*). Heteroimmune infection of φ80*dara* or λ*hy*80*dara* lysogens was used to test for late gene induction by the other phages. If the infecting phage can grow on the lysogen and induce the late genes of the prophage, arabinose isomerase will be synthesized, resulting from transcription of the arabinose operon by an RNA polymerase which initiated at the phage late gene promoter under control of the phage Q protein (*3*). The Q induced synthesis of arabinose isomerase from λ*hy*80*dara* has been shown to be independent of $N_\lambda$ (product of the λ N gene), an inducer of λ early protein, once $Q_\lambda$ is present (*3*). Figure 1 shows data leading to a similar conclusion, here λ*imm*$^{21}$ infected lysogens of λ*hy*80*dara* and φ80*dara*. The λ*imm*$^{21}$ was able to grow on both lysogens, but $Q_\lambda$ was able to induce only the late genes of λ. As λ*imm*$^{21}$ cannot complement a λ$N^-$, i.e., λ*imm*$^{21}$ does not possess an $N_\lambda$ (*4*, *5*); $N_\lambda$ is not directly required for inducing λ late genes.

Table 1 shows the ability of various lambdoid phages to induce arabinose isomerase from λ*hy*80*dara* and φ80*dara*. The first entries, λ, λ*imm*$^{434}$, and λ*vir*, are as expected; those phage able to grow in a λ lysogen but possessing $Q_\lambda$ induce isomerase from the phage with arabinose genes under control of a λ late gene promoter, λ*hy*80*dara*. The $Q_\lambda$ does not induce the arabinose operon on φ80*dara*, a result in agreement with the results of Szpirer and Brachet, who showed by other means that the $Q_\lambda$ does not induce the late genes of φ80 (*6*). Phages 381, 424, and 434*vir* do not induce λ or φ80 late genes. The same is seen for λ*imm*$^{434}$ *gin*$A_3$ (*7*) and λ*imm*$^{434}$p4, two phages apparently having replaced their Q gene with a Q-like gene and promoter from another prophage (*8*).

The inability of 434 to induce the late

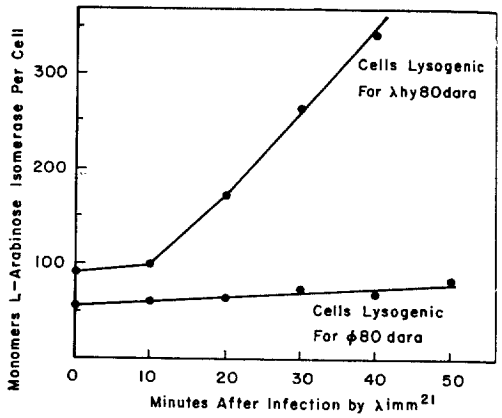


FIG. 1. Heteroimmune induction of λ and φ80 late genes by λ*imm*$^{21}$. For details of the heteroimmune infection, see (*3*). In brief, isogenic strains lysogenic for λ*hy*80*dara* and φ80*dara* were grown to 1–2 × $10^8$ ml in broth, concentrated 10- to 20-fold in 0.01 *M* $MgSO_4$, infected at a m.o.i. of 5 with the λ*imm*$^{21}$. After 20 min adsorption they were diluted into prewarmed broth and grown at 35C. Samples for isomerase assay were taken each 10 min for 40 min. At the same time the phage was titered on the two lysogens and their nonlysogenic parent. When phage is not added, but cells are otherwise treated as above, the basal level of 50–100 monomers per cell does not change during the 50 min.







| Phage[b] | Induction λ*hydara* lys |
|---|---|
| λ | <.1 |
| λ*vir* | 1 |
| λ*imm*$^{434}$ | 1 |
| λ*imm*$^{21}$ | 1 |
| 434*vir* | <.1 |
| 424 | <.1 |
| 381 | <.1 |
| λ*imm*$^{434}$*gin*A3 | <.1 |
| λ*imm*$^{434}$p4 | <.1 |
| φ80$C_{125}h_{434}$ | <.1 |
| φ80*vir* | <.1 |
| 81 | <.1 |
| 82 | <.1 |

[a] The procedure described in Fig. 1 stock as it was highly unstable.

[b] The λ and λ*vir* were from Mark P

[c] Induction of a lysogen is calculat tion relative to the slope following in

[d] In cross streaking a lysogen of λ*h* whereas φ81 weakly kills the cells.

genes of λ implies that it does no Q product identical to the lambd uct. This allows a refinement in t location of the Q gene on the λ ness, Doerfler, Egan, and Black mined that the Q gene is close to ever, DNA heteroduplexes betw 434 show homology from 87.3 t nonhomology from 90.7 to 94.7 ( sults showing $Q_\lambda \neq Q_{434}$ imply t part of the lambda Q gene lies to 90.7. This conclusion is subject t vation that a difference in biologi could be generated by base se parently homologous in electron observation of DNA heteroduple

Interestingly, phages φ81 and duce the late genes of φ80. Pha found in the same isolate of *E.* and therefore both phages could lar systems for inducing late gene ever, the induction of φ80 late g is less expected, since φ82 sho able homology to λ and may more closely related to λ than φ

It is possible that the induction

TABLE 1

THE HETEROIMMUNE INDUCTION AND PLATING EFFICIENCIES OF LAMBDOID PHAGE ON λ*hy*80*dara* AND φ80*dara* LYSOGENS[a]

| Phage[b] | Induction of λ*hydara* lysogen[c] | Plating efficiency on λ*hydara* lysogen relative to nonlysogen | Induction of φ80*dara* lysogen[c] | Plating efficiency on φ80*dara* lysogen relative to nonlysogen |
|---|---|---|---|---|
| λ | <.1 | .0 | <.1 | 1.1 |
| λ*vir* | 1 | 1.2 | <.1 | 1.0 |
| λ*imm*$^{434}$ | 1 | .9 | <.1 | .7 |
| λ*imm*$^{21}$ | 1 | .9 | <.1 | .9 |
| 434*vir* | <.1 | 1.0 | <.1 | .8 |
| 424 | <.1 | 1.0 | <.1 | 1.0 |
| 381 | <.1 | .6 | <.1 | .2 |
| λ*imm*$^{434}$*gin*A3 | <.1 | .8 | <.1 | .7 |
| λ*imm*$^{434}$p4 | <.1 | .9 | <.1 | 1.0 |
| φ80$C_{125}h_{434}$ | <.1 | .5 | <.1 | .0 |
| φ80*vir* | <.1 | <.001[d]! | 1 | 1.0 |
| 81 | <.1 | .0[d] | 1 | .9 |
| 82 | <.1 | .7 | .4 ± .2 | .4 |

[a] The procedure described in Fig. 1 was followed. Phage φ82 was used within 2 hr of making a plate stock as it was highly unstable.

[b] The λ and λ*vir* were from Mark Ptashne; the rest were from Ethan Signer via Ira Herskowitz.

[c] Induction of a lysogen is calculated from the slope of the isomerase accumulation following infection relative to the slope following infection of the λ*hydara* lysogen by λ*vir*.

[d] In cross streaking a lysogen of λ*hydara* against phage stocks at 1–5 × $10^9$/ml, φ80*vir* does nothing, whereas φ81 weakly kills the cells.

genes of λ implies that it does not possess a Q product identical to the lambda Q product. This allows a refinement in the possible location of the Q gene on the λ map. Hogness, Doerfler, Egan, and Black (*9*) determined that the Q gene is close to 90.0. However, DNA heteroduplexes between λ and 434 show homology from 87.3 to 90.7 and nonhomology from 90.7 to 94.7 (*10*). My results showing $Q_\lambda \neq Q_{434}$ imply that at least part of the lambda Q gene lies to the right of 90.7. This conclusion is subject to the reservation that a difference in biological activity could be generated by base sequences apparently homologous in electron microscopic observation of DNA heteroduplexes.

Interestingly, phages φ81 and 82 both induce the late genes of φ80. Phage φ81 was found in the same isolate of *E. coli* as φ80 and therefore both phages could have similar systems for inducing late genes (*11*); however, the induction of φ80 late genes by φ82 is less expected, since φ82 shows considerable homology to λ and may therefore be more closely related to λ than φ80 (*10*).

It is possible that the induction by φ81 and φ82 of the late genes of φ80 is a result not of $Q_{81}$ or $Q_{82}$ initiating an RNA polymerase at the late gene promoter, but simply from recombination between φ81 or φ82 and φ80. Such recombination would then place the arabinose genes on φ80 under direct control of φ81 or φ82. Without Q gene mutants in phage φ81 or 82, this possibility cannot be excluded; it seems unlikely though, since 434 did not detectably induce λ late genes, and yet λ and 434 possess extensive homology through most of their late gene regions (*10*).

These results show that phages φ81 and 82 are able to induce the late genes of φ80; whereas the other temperate coliphages tested are not able to turn on late genes of λ or φ80.

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## A Comparison of Certain
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Walters and Scott (*1*) isolated D
yellow mottle virus (DYMV) fro
*dium* spp. growing in central Arka
virus infected legumes only but rea
turnip yellow mosaic virus (TY
serum. Electron micrographs o
preparations showed "empty" a
particles. The work described here
certain additional properties of D
those of TYMV.

The DYMV isolate was passe
three local lesion transfers on a "
*Desmodium* sp. (*1*). Virus was pu
infected bean (*Phaseolus vulgaris*
Northern") harvested and frozen
after inoculation. Infected tissue w
enized in 0.2 *M* $NaH_2PO_4$ (1 g
and the homogenate squeezed
cheesecloth. The extract was adju
5.0 with 0.1 *N* HCl, stirred for
room temperature, and subjecte
alternate low speed (8000*g* for 10
high speed (80,000*g* for 90 min)
tions. Virus pellets were resus
0.01 *M* phosphate buffer, pH
TYMV, obtained from J. M. Ka
ville, Maryland, was purified fro
chinese cabbage (*Brassica chinens*
hili") by the same procedure.

The schlieren pattern obtained
fied DYMV preparations in t
Model E analytical ultracentrifug
cal of members of the Tymovirus
Two components with sedimenta
cients of about 54 S and 114 S, es
Markham's graphical method (*3*)
served (Fig. 1).

Antisera were developed in rab
peated intramuscular injections
viruses mixed with Freund's inco
juvant. Comparisons of the two
1 mg/ml using homologous and h
antisera in gel diffusion tests in
in water with 0.02% sodium azi
in reciprocal spur formation (Fi
actions of DYMV and TYMV wi