

Abortive Transduction of Resistance Factor by Bacteriophage P22 in *Salmonella typhimurium*

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When R factor 222 is transduced by bacteriophage P22 in *Salmonella typhimurium*, most recipient bacteria which adsorb transducing particles do not give rise to transductant clones (i.e., transduction is abortive); however the transduced drug-resistance genes can be rescued by recombination with the resistance-transfer factor or R factor carried by the recipient.

When bacteriophage P22 is used to transduce drug-resistance markers of f_i^+ R factor 222 (5) in *Salmonella typhimurium* LT-2, they segregate into a sulfanilamide (*sul*), streptomycin (*str*), and chloramphenicol (*cam*) component and a tetracycline (*tet*) component (3). A majority of these drug-resistant transductants are unable to transfer their drug-resistance markers by causing conjugation. The *tet* marker transduced by phage P22 in *S. typhimurium* appeared to be integrated into the bacterial chromosome near *pro*, i.e., at or near the P22 prophage attachment site; the transductants concerned were exceptional in that they behaved as defectively, instead of normally, lysogenic for P22 (1). The *sul*, *str*, and *cam* markers transduced by P22 also seemed to be integrated at a different site on *S. typhimurium* chromosome (Watanabe and Ogata, unpublished data). On the other hand, R factor 222 is genetically unstable in *S. typhimurium*, and the *sul*, *str*, and *cam* markers are often simultaneously lost, although the *tet* marker is much more stable (4). Any drug-resistance markers remaining after such spontaneous loss continue to be transmissible by conjugation. We have recently found that f_i^+ R factor 222-R₃, which is a spontaneous segregant carrying *sul*, *str*, and *cam* markers developed from 222, is also genetically unstable in *S. typhimurium* LT-2, simultaneously losing its three drug-resistance markers at high frequencies (Watanabe and Ogata, submitted for publication). It was thought of interest to study whether the resistance transfer factor (RTF), the episomal portion of R factor (2), still remains in these drug-sensitive segregants.

We have attempted a study on this point by using the drug-sensitive segregants obtained

from LT-2 (222-R₃) as recipients for transduction of R factor 222, in the expectation that the transduced drug-resistance markers (or defective R factors) might acquire conjugal transferability, if the drug-sensitive segregants still harbor RTF. The procedure for transduction was the same as that reported previously (3). Transduced drug-resistance markers were conjugally transferable in the case of one of the five, independently isolated drug-sensitive segregants used as transductional recipients (Table 1). It was also found that the frequencies of drug-resistant transductants were increased about 10 times in this recipient. These results seem to be best understood by supposing that this segregant retained RTF. In other words, the transduced drug-resistance markers of R factor 222 seem to have been rescued by recombining with RTF in about 90% of the transductants; otherwise, they would have become abortive (unless they were integrated into the host chromosome).

We then used LT-2 carrying 222-R₃ or 222-TC (a segregant R factor with the *tet* but not the *sul*, *str*, or *cam* markers derived from 222) as recipients for transduction of 222 by phage P22. The frequencies of drug-resistant transductants were again increased about 10 times by the presence of these R factors in the recipient and, furthermore, all of the drug-resistant transductants obtained had the ability to transfer their drug-resistance markers conjugally. It is interesting to note that a transductant with the *tet* but without the *sul*, *str*, and *cam* markers was obtained in the transduction of 222 to a recipient carrying 222-R₃, and that this *tet* marker was transmissible. This transmissible *tet* R factor must have developed as a result of genetic recom-

TABLE 1. Transduction of f_i^+ R factor 222 (*sul*, *str*, *cam*, *tet*) by phage P22 to various derivatives of *Salmonella typhimurium* LT-2

Recipient	Selected marker	Frequency of transductants ^a	Resistance marker(s) of transductants	f_i^+ Transductants ^b
LT-2 (R ⁻)	<i>cam</i>	3.5×10^{-6}	<i>cam</i> , <i>sul</i> , <i>str</i>	0/50
	<i>tet</i>	2.6×10^{-7}	<i>tet</i>	0/50
Sensitive segregants ^c				
No. 1	<i>cam</i>	3.8×10^{-6}	<i>cam</i> , <i>sul</i> , <i>str</i>	0/50
	<i>tet</i>	2.2×10^{-7}	<i>tet</i>	0/50
No. 2	<i>cam</i>	3.5×10^{-6}	<i>cam</i> , <i>sul</i> , <i>str</i>	0/50
	<i>tet</i>	3.0×10^{-7}	<i>tet</i>	0/50
No. 3	<i>cam</i>	3.9×10^{-6}	<i>cam</i> , <i>sul</i> , <i>str</i>	0/50
	<i>tet</i>	2.2×10^{-7}	<i>tet</i>	0/50
No. 4	<i>cam</i>	3.3×10^{-6}	<i>cam</i> , <i>sul</i> , <i>str</i>	91/100
	<i>tet</i>	3.0×10^{-6}	<i>tet</i>	90/100
No. 5	<i>cam</i>	3.9×10^{-6}	<i>cam</i> , <i>sul</i> , <i>str</i>	0/50
	<i>tet</i>	2.9×10^{-7}	<i>tet</i>	0/50
LT-2 (222-TC) ^d	<i>cam</i>	1.2×10^{-5}	<i>cam</i> , <i>sul</i> , <i>str</i> , <i>tet</i>	10/10
LT-2 (222-R ₃) ^e	<i>tet</i>	3.5×10^{-6}	<i>tet</i> , <i>cam</i> , <i>sul</i> , <i>str</i>	9/9
			<i>tet</i>	1/1

^a Frequency of transductants per plaque-forming unit. Multiplicity of infection was about 5.

^b Transductants with ability to transfer their drug-resistance markers by conjugation.

^c Drug-sensitive segregants which developed spontaneously from LT-2 (222-R₃).

^d Spontaneous segregant carrying R factor (*tet*) which developed from LT-2 (222).

^e Spontaneous segregant carrying R factor (*sul*, *str*, *cam*) which developed from LT-2 (222).

bination between the transduced *tet* marker and the R factor 222-R₃ present in the recipient. These results suggest that the abortively transduced drug-resistance markers were rescued by recombination with R factors in the recipient.

Our previous investigation on the transductional superinfection with R factors did not show any increase in the transduction frequencies, even when the R factors in the donor and recipient were both of f_i^+ type (6). The discrepancy between the present and previous results could be explained as follows. In the earlier experiments we used only R factors of independent origins, whereas we have employed R factors of the same origin in the present investigation. Thus, we may conclude that homologies may be lower among independent isolates of R factors even when they are of f_i^+ type than among the R factors of the same origin.

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