I'm writing a reprint J.C.

"On August 18, 1988 I had the opportunity of visiting Dr. Victor J. Freeman at his home in Millbrae, Calif. He is a retired psychiatrist, having left laboratory microbiology for a career in public health, and then psychiatry. Discussing the steps that led to his discovery of lysogenic conversion, we perused some of his old files, and there is a very clear story in one of his reports to NIH. Having a collection of phages active on non- exotoxin-producing strains of Corynebacterium, he used the phage to lyse those cells in hopes of releasing an endotoxin different from the canonical diptheria exotoxin.

On page 4 of his report (August 3, 1951) he refers to phage B studies; the relevant paragraph is:

'While carrying on investigations with phage B it was discovered that this phage had a marked activity on several avian cultures of C. diphtheriae. In view of this result it was decided to test for the possibility of the presence of a dermal necrotic endotoxin after the method of Lazarus and Gunnarson, J. Bact. 1947, 53:705-714. The phage lysates of the avian cultures did produce a dermal factor toxic for guinea pigs. Further investigations soon revealed that the toxic substance being produced was true diphtheria toxin. In view of these findings the principal emphasis of the research project was modified to take advantage of these very pertinent observations etc. There was no practical development of a phage typing system. Several interesting patterns of specific lysis were developed through phage adaptation but owing to this instability these tests could not be utilized for typing purposes.' "

An equally productive anticipation can be seen in Frobisher & Brown's work in 1927 [21]: they had the idea that scarlatina was caused by a virus only secondarily related to the streptococcus. They evidently did induce a (lysogenic?, but unstable) conversion of a "cheese strep" to an erythrogenic one with filtrates. Their work was repeated by Bingel 1947, and taken up again by John Zabriskie 20 years after that. Finally, Ferretti's group in Oklahoma have recently put the finishing touches on the demonstration that this is another canonical lysogenic conversion, the toxin genes having been located on the phage (plasmid). [18].

In 1953, I was collaborating with P.R. Edwards on the serotypy of the H antigens of Salmonella [33]. He told me of the conversion of some of the O somatic types with antisera. I tried to persuade him that antibody was a very unlikely reagent for transformation, he was just seeing another example of selection. Nevertheless, as Zinder and I had just demonstrated phage- mediated transduction, was there possibly some phage in his antisera? these were, after all, purified by repeated "absorption" with heterologous bacteria. He countered that there was probably dissolved antigen there [5]; perhaps that was being taken up by the altered bacteria. It turn out, indeed there was phage! -- Iseki and Sakai, 1953, [29] in the first of many such examples, demonstrated the modulation of somatic antigen specificity in Salmonella by lysogenic conversion. That is, the phage genome includes determinants altering its host's development.