LEDERBERG, ESTHER M., University of Wisconsin, Madison, Wis.--Fine structure of the Gal loci in Escherichia coli K-12.--A distinctive recon (one of a group of closely linked loci separable by recombination) has been assigned to each of ten independently occurring Gal^- (galactose non-fermenting) mutants. All the mutants were obtained after UV irradiation except Gal_3 which was spontaneous. Allelism was tested by large scale matings and by transduction analysis.--Prophage-linked transduction via HFT λ, where practically every phage particle may result in a transductional event, produced heterogenotic clones when donor and recipient bore distinctive recons. The heterogenotes were unstable, segregating the two input and two crossover classes (Morse, Lederberg and Lederberg 1956). Two cistrons (cis-trans position effect groups) had been demonstrated: trans heterogenotes with members of the same cistron are phenotypically mutant. Galactokinase was missing in mutants of one cistron (Gal 2,8) while UDP transferase activity was absent in the other (Gal 1, 4, 6, 7), a defect corresponding to human congenital galactosemia, (Kalckar, Kurahashi 1957). Gal_3^- and Gal_9^- are genetically cistronic with <u>both</u> of the foregoing groups; they have not yet been successfully analysed for their enzymatic defect. Among two hundred new mutants, no recons identical with the first ten have recurred. Most have been assigned to either of the first two cistronic groups, but a small number which would represent a fourth genetic group have given only normal galactose-positive heterogenotes with every standard Gal^- tested. A few mutants were not transformable by λ but were found to carry other modifiers including that of hexose metabolism which obscure their relationship to the Gal^- group.--An attempt was made to map the Gal markers in linear sequence by their relationship to the Lp locus. Intercrosses of Hfr $M^- Gal_x^-$ x $F^- Gal_y^-$ were made on a medium which selected efficiently for $M^+Gal_x^+Gal_y^+$ recombinants, and these were then scored for the segregation of an Lp marker. However, the linkage of Gal-Lp proved to be not close enough to compensate for the perturbations of segmental loss from the Hfr parent, and the data were indecisive.

10th International Congress of Genetics, Montreal, 1958.