and treated with 'Antrycide'. The rate of disappearance of parasitæmia with the splenectomized group was also much prolonged as compared to the unsplenectomized ones.

The above results suggest that the reticulo-endothelial system participates in the chemotherapeutic activity of 'Antrycide'. Its action is similar to trypanocidal drugs like suramin in this respect, as the activity of the latter has been found to be bound up with the reticulo-endothelial system[2]. But the exact mode of participation of the reticulo-endothelial system, that is, (1) whether the drug acts chiefly in stimulating the immunity, or (2) indirectly kills the organism by an opsonin-like reaction, or (3) facilitates a longer drug–parasite contact, is still not clearly understood. Work is in progress to explore the above possibilities.

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Resistance to Colicin *E* as a Genetic Marker in *E. coli* K12

As many genetic markers as possible are needed for the elucidation of possible mechanisms by which bacterial recombination occurs.

A whole group of potential markers is available in the colicins. These are antibiotics produced by some *E. coli* strains, which can inhibit the growth of other strains of the same or closely related species[1]. Unfortunately, none of these substances has been obtained pure, so that they cannot be used as selective agents during the recombination technique but only as unselected markers with which the parent strains and their progeny can be labelled. Several of the colicins produce wide zones of inhibition with sensitive strains and are therefore possible useful markers[2]. In particular, colicin *E* possesses all the requirements of a good marker and its linkage to other known markers can be studied.

The strains of *E. coli* used were the wild-type *K*12 strain which grows on inorganic salt medium with glucose (minimal medium)[3], strain 58.161 requiring methionine (M–) for growth, and strain *W*677 requiring threonine (T–), leucine (L–) and thiamine (B_1–) for growth; both *F*+ and *F*− types of these strains were used. All these strains are sensitive to colicin *E*, and resistant mutants were selected by inoculating approximately 10^9 cells of the parent strain on to nutrient agar plates containing 10 per cent of a 24 hr. broth filtrate of the colicin *E*-producing strain *CA*38. Recombinations were carried out and the progeny tested by the techniques described previously[4]. Colicin *E* sensitivity of the recombinants was tested by the method of Fredericq[5].

The results of four recombination experiments in which the mixture of the washed parent strains was seeded on to different selective media are shown in Table 1. In the first two crosses, in which *W*677 was the *F*− parent, selection for T+, L+ and B_1+ by seeding the mixture on to unsupplemented minimal medium yields a progeny of which 75–82 per cent is resistant to colicin *E*. Addition of thiamine to the selecting medium, which allows B_1-dependent colonies to grow, greatly increases the number of recombinants, and of these only 2–14 per cent are resistant to colicin *E*. There is evidently an association or linkage between the B_1+ and E^R characters of the *F*+ parent. The cross of *W*677 $E^R/F-$ with 58.161/*F*+, in which the *F*− parent carries the marker for colicin *E* resistance, gives results which are in agreement with the localization of the E^R locus close to that of B_1. In this case, when selection is for T+, L+ and B_1+ the locus for colicin *E* sensitivity is present in 50 per cent of the progeny. Removal of selection for B_1+ reduces this to 6 per cent, that is, 94 per cent of the progeny have the E^R locus of the *F*− parent. The cross of $WE^R/F+$ with 58.161/*F*− also yielded concordant results; in this instance, selection for *M*+ carries over 56 per cent of colicin E^R; addition of B_1 to the selecting medium gives a progeny 90 per cent of which is E^R. The loci for M and B_1 in 58.161 are known to be closely linked[6], and the greatly increased transfer of E^R when survival of the B_1− marker of 58.161 is allowed shows that the locus for colicin E^R lies between the loci for M and B_1.

Table 1. Analysis of Recombinants

Cross	Supplements to minimal medium	Recombinants (Nos.)	% B_1	% colicin E^R
58 E^R × *W*/*F*−	None	88	—	75
	B_1	600	55	2
	Lactose	50	—	66
	Lactose B_1	200	52	14
*K*12 E^R × *W*/*F*−	Strep.	500	—	82
	Strep. B_1	800	70	14
	Strep. lactose	400	—	92
	Strep. B_1 lactose	600	96	24
58/*F*+ × *W* E^R/*F*−	None	100	—	50
	B_1	600	?	94
	Lactose	50	—	58
	B_1 lactose	200	?	93
W E^R/*F*+ × 58/*F*−	None	150	—	56
	B_1	175	?	90

The cross of $WE^R/F+$ with 58.161/*F*− also yielded concordant results; in this instance, selection for *M*+ carries over 56 per cent of colicin E^R; addition of B_1 to the selecting medium gives a progeny 90 per cent of which is E^R. The loci for M and B_1 in 58.161 are known to be closely linked[6], and the greatly increased transfer of E^R when survival of the B_1− marker of 58.161 is allowed shows that the locus for colicin E^R lies between the loci for M and B_1.

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A *Limax*-Amœba from the Rectum of the Grass Snake, *Natrix natrix*, as a Facultative Aerobe *in vitro*

Although small amœbæ of the *limax*-type appear fairly commonly in cultures initiated from standing fæces or the intestinal contents of animals, in only a very few cases has the truly entozoic status of the amœba thus recovered been authenticated. The test hitherto has been the direct observation of the trophic amœbæ in host material, either in fresh