wing movements. From our film we could not positively associate a particular wing movement with either of the two pulses. Twenty independent analyses of the five filmed type IV phonatomes showed these apparent movements during the first pulse: opening, $n = 7$; closing, $n = 1$; no movement, $n = 12$. Corresponding figures for apparent movements during the second pulse were as follows: opening, $n = 2$; closing, $n = 14$; no movement, $n = 4$. The first pulse is probably produced by an opening movement and the second by a closing.

The transitions from type I to type II and from type II to type III phonatomes are distinctive. The sequence of type I phonatomes ends with several that are variable (Fig. 1C; two films; two individuals). The amplitude of wing movement is greater (suggesting type II), the period becomes shorter (suggesting type III), and sometimes opening or a portion of closing is silent. Sometimes a group of such erratic type I phonatomes is followed by a short sequence of regular ones, that gives way again to erratic type I and then to type II phonatomes. Perhaps the katydid successfully shifts to type II phonatomes after an initial failure. The transition from type II to type III phonatomes is sudden (Fig. 1, C and D; four films; four individuals). The final type II phonatome has a slightly prolonged hold during closing, and the initial type III phonatome has a closing sound that begins gradually (as in type II phonatomes).

The complexity of wing movements in this species far exceeds any previously described. Those working with simple movements in one- or two-phonatome species (*2–4*) should note the challenges that remain. Systematists should note that wing-movement cycles provide an important new clue to homology and analogy among signals (*10*). Those taxonomists who have assumed that the distinctive features of the calling song can be deduced from features of the stridulatory apparatus (*11*) should note that four phonatomes come from a single apparatus and that the variety of distinctive calling songs one such apparatus might produce by changes in the sequence and timing of the four phonatomes would easily exceed the number of species of katydids.

The structure of the stridulatory file of Uhler's katydid merits comment. For instance, one might expect that the file would show some specialization facilitating the two-step closures of type I and type II phonatomes. In fact, certain species of Phaneropterinae (the subfamily including Uhler's katydid) have the most complex files known: some files have sharp bends, and others have sudden transitions in the structure and spacing of the teeth (*12*). However, the file of Uhler's katydid is remarkably ordinary—a row of nearly uniform teeth gently curving at either end (see cover). The calling songs of phaneropterines with complex files are unknown.

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Galactokinase: Evidence for a New Racial Polymorphism

Abstract. *Activities of galactokinase and galactose-1-phosphate uridyltransferase in red cells were assayed in a mixed racial population of 645 pregnant women. The distribution of individual transferase activities for black subjects was the same as that for whites. In contrast, the distributions of individual galactokinase activities differed significantly in blacks and whites, the mean for the black population being 30 percent lower than the mean for the white population. The same racial difference was found when red cell galactokinase activity was examined in males and in newborns. Because low-, intermediate-, and high-galactokinase activities appear to segregate within several black families, this observed difference suggests a new, racially determined enzyme polymorphism.*

Galactokinase and galactose-1-phosphate uridyltransferase are essential for the utilization of galactose in man. Deficiency of each of these enzymes is associated with an autosomal recessive inborn error in metabolism known to cause a clinically significant disorder (*1*). Galactokinase deficiency is characterized by juvenile cataracts. Transferase deficiency, or "classical" galactosemia, is associated by hepatosplenomegaly, cataracts, and failure to thrive. Heterozygotes for either of these enzyme deficiency diseases were once thought to be easily distinguished by quantitative assay of red cell enzyme activity, because they have half the normal enzyme activity. The identification of the Duarte variant of human transferase (*2*), an allele that produces about half the activity of the wild type allele, made it necessary to supplement quantitative enzyme data with appropriate family studies or electrophoresis or both techniques (*3*) in order to establish transferase genotypes. The results

reported here suggest that similar criteria may be necessary to identify the genotype of carriers for galactokinase deficiency, particularly in the black population.

Hemolyzates were prepared from heparin-treated blood by washing the red cells three times with isotonic saline. One volume of packed red cells was diluted with three volumes of 13.3 m*M* phosphate buffer at *p*H 7.0, containing 6.6 m*M* dithiothreitol and 0.8 m*M* ethylenediaminetetraacetic acid. After freezing and thawing, 50-μl portions were used to determine galactokinase activity and transferase activity as described (*4*).

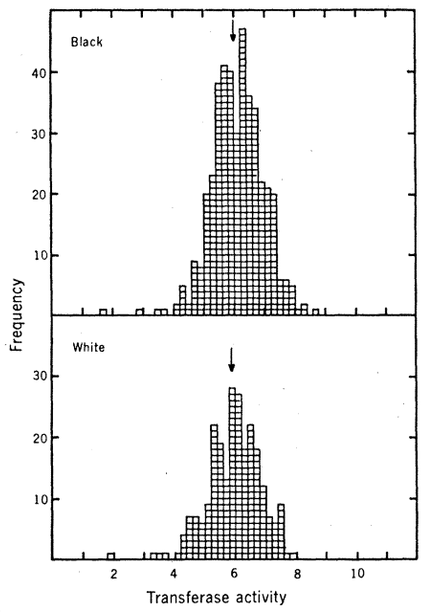
The distributions of individual transferase activities for pregnant females (Fig. 1) showed no obvious difference between blood samples from black and white subjects. The mean for 216 whites was 5.85 (standard deviation = 1.18, standard error = 0.080), and that for 429 blacks was 6.00 (S.D. = 1.17, S.E. = 0.056).

The distributions of individual galactokinase activities for pregnant females (Fig. 2), however, demonstrated a significant difference between black and white racial groups. The mean for 216 whites was 0.302 (S.D. = 0.127, S.E. = 0.009) and that for 429 blacks was 0.219 (S.D. = 0.130, S.E. = 0.006, $P < .0005$).

Hemoglobin concentration did not significantly influence galactokinase activity ($r = .057$ for blacks, .110 for whites), and there was no significant influence of gestational age on galactokinase activity ($r = -.142$ for blacks, $-.199$ for whites). These observations tend to minimize nongenetic factors as an explanation for the observed racial difference.

The possibility that sex or pregnancy might have some effect on galactokinase activity was tested by examining enzyme activity in 42 males. The mean for 19 blacks was 0.228 (S.D. = 0.062, S.E. = 0.014), and that for 23 whites was 0.359 (S.D. = 0.093, S.E. = 0.019); these data demonstrate the same significant difference found for the pregnant females.

Galactokinase assays of umbilical cord blood also demonstrate that blacks had less enzyme activity than did whites, even though enzyme activity in cord blood was three to four times that found in adults. The mean for 33 blacks was 0.933 (S.D. = 0.260, S.E. = 0.045), and that for 11 whites was 1.160 (S.D. = 0.129, S.E. = 0.039). No racial differences were seen in red cell transferase activity in either the male or newborn groups.



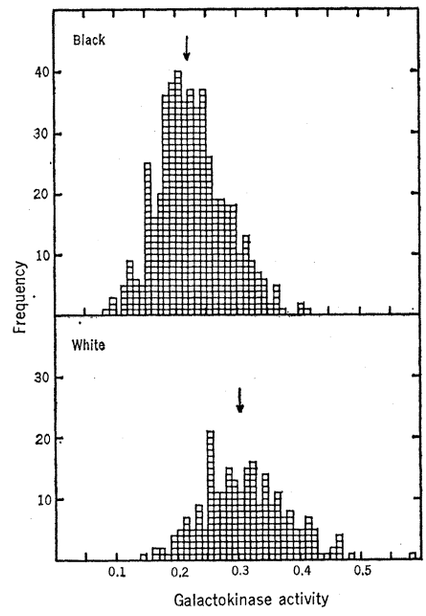


Fig. 1 (left). The distribution of individual galactose-1-phosphate uridyltransferase activities in black and white pregnant females. The arrows indicate the population means. Enzyme activity is expressed as micromoles of substrate converted per hour per milliliter of red cells. Fig. 2 (right). The distribution of individual galactokinase activities in black and white pregnant females. The arrows indicate the population means. Enzyme activity is given as micromoles of substrate converted per hour per milliliter of red cells.

Because both enzyme assays were performed on the same hemolyzate from each individual and no racial difference was found for transferase activity in any of the groups tested, the difference described for galactokinase activity seems to be specific. There is currently no evidence that galactokinase deficiency is more common in the black race, so it seems unlikely that this observed racial difference can be explained by a high frequency of the galactokinase deficiency gene among blacks. In our initial family studies, we have identified families in which activity of red cell galactokinase in siblings and parents falls into three ranges: (i) "high," similar to that of the white population we have studied, (ii) "low," approximately 30 percent of the "high" activity, and (iii) "intermediate," with activities approximately midway between "high" and "low."

Critical segregation data is needed to support our postulation that there is a common polymorphic gene affecting red cell galactokinase activity in the black race. Several polymorphisms have been reported to be more common in certain racial or ethnic groups. Reed has described several genetic loci in which variants are so characteristic of one racial group that they are useful for estimating white ancestry in American blacks (*5*).

The high incidence of lactase deficiency in blacks as compared to whites (*6*) represents a similar racial difference. It would be interesting to know whether lactase deficiency in blacks has to some extent tended to prevent clinical manifestations of the disease or to protect galactokinase variants from selective pressure.

The results reported here indicate that data from obligate heterozygotes (parents of individuals with galactokinase deficiency) in the white population cannot be used to estimate the heterozygote frequency of galactokinase deficiency in black populations. In our random population more blacks than whites had apparent heterozygote levels of galactokinase activity. We suspect that this difference may be due to a galactokinase allele or alleles (a polymorphism), more common in the black population, that produces lowered red cell galactokinase activity and is distinct from the allele that causes, in the homozygous state, the disease now recognized as galactokinase deficiency.

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The Cutaneous "Rabbit": A Perceptual Illusion

Abstract. *Anomalous localizations of mechanical and electrical cutaneous pulses are produced when widely separated bodily points are successively stimulated with trains of taps. The observer experiences a manifold of discrete "phantom" impressions connecting the points actually touched. The theoretical basis for this perceptual phenomenon is not understood, but some boundary conditions are specified.*

In the course of designing some experiments on the cutaneous perception of mechanical pulses delivered to the back of the forearm, it was discovered that, under some conditions of timing, the taps produced seemed not to be properly localized under the contactors. For example, if five brief pulses (2-msec duration each, separated by 40 to 80 msec) are delivered to one locus just proximal to the wrist, and then, without break in the regularity of the train, five more are given at a locus 10 cm centrad, and then another five are added at a point 10 cm proximal to the second and near the elbow, the successive taps will not be felt at three loci only. They will seem to be distributed, with more or less uniform spacing, from the region of the first contactor to that of the third. There is a smooth progression of jumps up the arm, as if a tiny rabbit were hopping from wrist to elbow. If the original timing is retained and the number of taps (N) at each locus is reduced, the hops get longer; if N is increased (up to a limit), the hops become shorter.

An adequate explanation of this striking phenomenon is not yet at hand. Several hypotheses have led to interesting experiments and, in a few instances, to crucial ones that have destroyed the hypotheses. It is thus possible to identify with certainty some principles not at work; it is more difficult to demonstrate vital contingent conditions.

The experimental arrangements should be described briefly. Two or more contactors, made of short lengths of Lucite rod (0.6 cm diameter) slightly rounded at the tips and mounted rigidly on the free ends of Clevite bimorph benders, are driven by trains of square-wave pulses from a system comprising a Tektronix 162 waveform generator, a 161 pulse generator, and a Langevin power amplifier. This combination permits a pulse of constant duration (2 msec) but with interpulse durations varying over a wide range. Intermediate between the bimorphs and the electrical driving system is a Tally tape reader. Eight-channel tapes may be prepared with any desired program of pulses; N, interstimulus interval (ISI), and overall duration can be made to vary independently of each other. In our experiments the contactors, two to five in number, rested on the skin with an initial static pressure of 15 g. The forearm rested on foam rubber and was immobilized with small sandbags.

Several interrelated questions arise at once. Is the rabbit effect an accident of the direction in which the contactors are successively energized? Is distance important? Is the ISI crucial? How does the effect vary with N? Does irregularity of pulsing alter the result? Are repetitions of a given sequence necessary to induce the effect?

Direction of sequence is not a vital matter; hopping can go down the arm as well as up it. Indeed, it is possible to have hopping in both directions at once. Five contactors were set on the forearm in lineal array. Contactor 3 was first energized, then contactors 2 and 4 (simultaneously), then 1 and 5 (also simultaneously), five successive pulses being delivered to each. The reverse (a "rabbit" collision) occurred when the sequence was reversed. It would be interesting to investigate other possible collision courses, since there is typically the impression that the taps extend beyond the terminal contactor.

Hopping has been observed when two contactors are as close together as 2 cm and as far apart as 35 cm. In one experiment it was possible to induce a vivid hopping that traveled up one arm, across the neck, and down the other arm by the use of five contactors, two widely separated on each arm and one on the nape of the neck.

The time between taps is not very critical; good, well-spaced hopping occurs over a wide range of ISI values. The first incipient stirrings from under the contactors—what we have dubbed the "threshold of exodus"—occurs with an ISI of about 200 msec. Two observers gave exodus thresholds (method of constant stimuli) of 193 and 205 msec, respectively. As ISI is shortened, N remaining constant at 5, there is wider apparent straying from the contactor locus, and at about 100 msec the hops are becoming somewhat evenly spaced. With further reduction of the ISI the trains of taps become faster until an ISI is reached (40 to 60 msec) for which the hopping is optimal in regularity and vividness. With further shortening of ISI, the perceived N becomes illusory. The 15 taps delivered to the three contactors may seem to be only six when the ISI is 20 msec.

The role played by N is not very important so long as not too many pulses are delivered at one locus. An N of 2 is adequate, though the effect is better with N of between 4 and 6. An N of 18 is too large; the taps tend then to get "anchored" under the contactor, and hopping does not occur. The effect occurs when N is 12, but less impressively. Probably N and ISI are interrelated; a systematic experiment would be needed to establish the facts.

Irregularity of pulsing disturbs the rabbit effect. With four pulses on each of three contactors the omission of a single pulse between trains does not destroy the effect but reduces its distinctness; faster overall presentation rates are then required to preserve the effect. Insertion of two blank intervals brings about an irregular rhythm at some speeds. Three and four blanks seem impossible to bridge; the effect is then totally destroyed. The interrelations among N, ISI, and gap length would be worth investigating.