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Chromosomal Polymorphism in the White-Throated Sparrow, Zonotrichia albicollis (Gmelin)

Abstract. *In a study of 35 white-throated sparrows five distinct karyotypes were observed. A chromosomal polymorphism is present which involves at least two pairs of macrochromosomes. This species is phenotypically polymorphic with selective breeding occurring between morphs. Phenotype is related to chromosomal constitution, and selective breeding appears to maintain heterozygosity within the population.*

Distinct chromosomal polymorphism is rare in the subphylum Vertebrata (*1*), and none has previously been recorded in the class Aves. This report describes the chromosomal polymorphism found in the white-throated sparrow in Ontario.

The chromosomal complements of 35 individuals were obtained by growing, in primary culture (*2*), cells from kidneys, whole embryos, and feather pulp.

This sparrow has a typically avian karyotype containing 82 or 84 chromosomes. Easily recognizable macrochromosomes grade into small microchromosomes, the smallest of which are difficult to count even in good preparations. Chromosome 12, a small metacentric, is a useful marker; the chromosomes following it appear acrocentric, with the exception of pair 18, which is metacentric.

Figure 1 shows the first 24 chromosomes in the karyotypes of four individuals. In all the birds studied, the first pair of chromosomes is homomorphic and has a submedian centromere. However, the chromosomes making up the next four elements in the karyotype may vary. Three single chromosomes of equal lengths appear to be involved in this variation; they have arbitrarily been designated as 2, 3, and M. Chromosome 2 has a subterminal centromere, chromosome 3 is acrocentric, and chromosome M is almost strictly mediocentric. Five combinations of these chromosomes have been observed: 2–2–3–3 (bird A and 15 others), 2–3–3–M (bird B and 14 others), 2–2–3–M (bird C and two others), 2–2–2–M (only bird D), and 2–2–2–3 (only one bird, not illustrated). Chromosome 4 is the Z chromosome in this species; in females (birds A and B) a distinct W chromosome is present. The remaining macrochromosomes are common to all the birds studied.

There is no apparent relation between sex and the number of chromosomes 2, 3, and M that are present. Since chromosomes 2, 3, and M are of equal length and since they always total four in number, it appears probable that basically only two pairs of chromosomes are represented and that rearrangements, including pericentric inversions, may have caused the observed polymorphism. Birds of type A (2–2–3–3) may represent the original karyotype of this species. Autosomal polymorphism due to pericentric inversions has recently been reported in *Peromyscus maniculatus* (*3*) and in *Mastomys natalensis* (1). Studies on the pairing of homologs at meiosis will be necessary, however, before the true nature of the polymorphism in the white-throated sparrow will be known. In particular these studies should show whether the apparent nullisomy (3 and M), monosomy (2, 3, and M), and trisomy (2) are real.

The white-throated sparrow has been described on the basis of external morphology as a dimorphic species with selective breeding occurring between the morphs; it was also suggested that this breeding system might ensure heterosis in populations of the species (*4*). The exact relationship between the phenotype and the chromosomal constitution of the morphs is unknown. All birds bright in nuptial plumage have a single M chromosome and all birds dull in nuptial plumage lack this chromosome.

While it now appears that more than two morphs exist, there is no doubt that bright birds of either sex always mate with duller birds. Since in the wild the bright morphs never mate with other bright morphs, one would expect few, if any, animals to be homozygous for chromosome M. None has been found so far.

Fertile eggs can now be obtained in the laboratory, and karyotypic studies of family units as well as wild sibs should yield more refined data on this unique system of polymorphism.

In the white-throated sparrow, morphological variation, assortative mating,

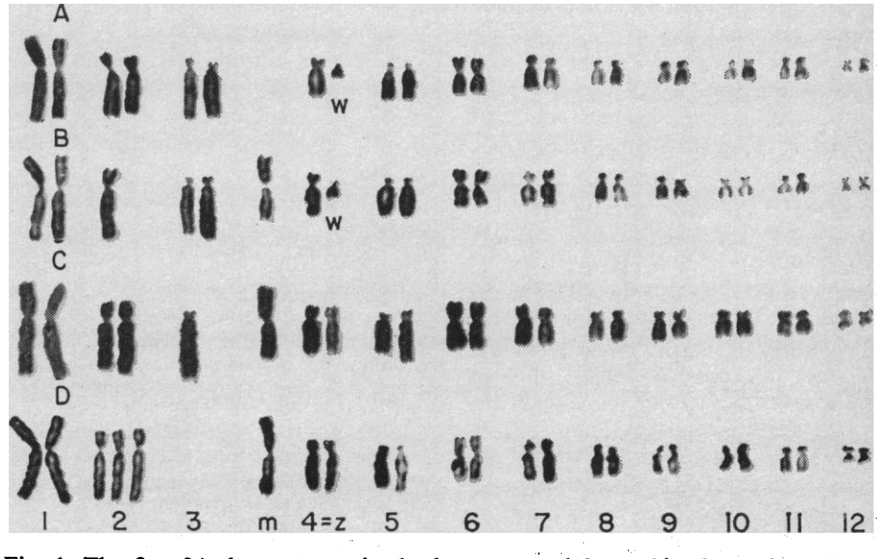

Fig. 1. The first 24 chromosomes in the karyotypes of four white-throated sparrows.

and chromosomal polymorphism appear to combine in an interlocking system to ensure heterozygosity. Perhaps this system permits the species to enjoy hybrid vigor while allowing it to avoid the penalty of large numbers of inviable gametes or zygotes.

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Control of the Activity of Escherichia coli Carbamoyl Phosphate Synthetase by Antagonistic Allosteric Effectors

Abstract. *The synthesis of carbamoyl phosphate required in both arginine and pyrimidine biosyntheses is carried out by a single enzyme in* Escherichia coli. *Opposed effects of pyrimidine nucleotides and of ornithine on the activity of the enzyme ensure a proper supply of carbamoyl phosphate according to the needs of the two biosynthetic sequences.*

Several regulatory patterns for single enzymes supplying divergent metabolic pathways with a common precursor have been encountered so far. For instance, control mechanisms such as multivalent repression (*1*), concerted feedback inhibition (*2*), cumulative feedback inhibition (*3*), or specific reversal of feedback inhibition (*4*) provide effective means of avoiding the regulatory interactions which otherwise could arise from such situations.

The regulation of the enzymic system which supplies carbamoyl phosphate (CP) for the synthesis of arginine and the pyrimidines in *Escherichia coli* is the subject of my report. Although this double function of CP has been known (*5*), definitive knowledge of the mechanism of its formation came only recently with the discovery, first in mushrooms (*6*) and later in *E. coli* (*7*), of an enzyme, glutamino-carbamoyl phosphate synthetase, which uses glutamine as the carbamoyl nitrogen donor (*8*). There is evidence that in *E. coli* a single glutamino-carbamoyl phosphate synthetase provides CP for both the arginine and pyrimidine pathways (*9*). The synthesis of the aforesaid enzyme is cumulatively repressed by the end products of the two pathways while its activity is subject to feedback inhibition by a pyrimidine nucleotide, uridine-5′-monophosphate (UMP).

Under the conditions used, the inhibition was no greater than 60 percent, even for UMP concentrations exceeding $10^{-2}M$. This partial feedback inhibition by UMP was considered essential in order to allow for the possibility of producing carbamoyl phosphate necessary for the biosynthesis of arginine. Similar cases of partial inhibition have been observed in the control of homoserine dehydrogenase from *E. coli* and *Rhodospirillum rubrum* (*10*). I now present a more complete picture of the control of the activity of glutamino-carbamoyl phosphate synthetase in *E. coli* based on a heretofore overlooked involvement of ornithine in that control.

Glutamino-carbamoyl phosphate synthetase was previously assayed by an indirect method based on the coupling of the synthesis of CP with ornithine-carbamoyl phosphate transferase (OCT), the citrulline formed being estimated colorimetrically (*7*). However, a direct method was used in my work. It involves accumulation of CP during a main incubation, followed by its conversion into citrulline by a short additional incubation in the presence of an excess of ornithine and OCT (legend to Fig. 1). This method avoids the constant presence of ornithine during CP synthesis, and its use enables one to study the influence of this amino acid on the activity of the synthetase. The direct assay, if one takes into account the chemical decomposition of CP and a slight activation of the enzyme by ornithine, gives the same results as the coupled assay.

Under these conditions, the enzyme appears much more sensitive to UMP inhibition, which is close to 100 percent for $2.5 \times 10^{-3}M$ UMP (Fig. 1). Ornithine, while slightly increasing the activity when taken individually, reduces considerably the efficiency of UMP as an inhibitor of the enzyme (Fig. 1). This effect of ornithine is obtained under conditions where it does not participate in the removal of CP through coupling with OCT. Ornithine may thus be seen as an allosteric effector of glutamino-carbamoyl phosphate synthetase which is responsible for the previously observed limitation of the feedback inhibition by UMP.

The study of the specificity of the feedback inhibition has shown that, although UMP is the most potent negative effector of the aforesaid synthetase, other pyrimidine nucleotides share this property. In decreasing order of effectiveness, uridine diphosphate, uridine triphosphate, cytidine monophosphate, cytidine triphosphate, and cytidine diphosphate are inhibitors of the enzyme but all are antagonized by ornithine in this effect (Table 1). The activity of the synthetase is thus un-

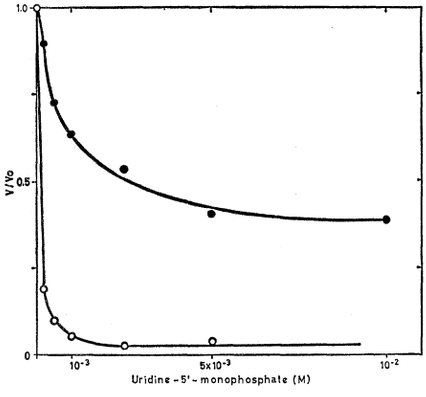

Fig. 1. Influence of ornithine on the feedback inhibition of *E. coli* glutamino-carbamoyl phosphate synthetase by UMP. This enzyme was extracted and partially purified (*8*), from the strain P4X of *E. coli* K12. The cells were grown on minimal medium No. 132 (*16*). The reaction mixture for the assay of the enzyme contained: $KHCO_3$, 30 μmole; ATP, 12 μmole; $MgCl_2$, 12 μmole; glutamine, 12 μmole; phosphate buffer (*p*H 7.5), 100 μmole; and enzyme in a total volume of 1 ml. This mixture was incubated for 15 minutes at 37°C. At this point, 1500 units of partially purified OCT from *E. coli* and 6 μmole of ornithine were added. The incubation was continued for 2 minutes in order to convert CP into citrulline. The reaction was stopped, and citrulline was determined (*7*). The activities obtained were corrected for the amount of CP formed during the additional 2-minute incubation period in the presence of ornithine and OCT. Solid circles, the reaction mixture contained ornithine from the start of the incubation; OCT was added after a 15-minute incubation period. Open circles, ornithine and OCT were added after 15 minutes incubation.