have not been adequately described or illustrated nor have their internal structure and connections been examined. Dorn's paper seems to have been virtually lost in the literature. I happened across it myself only after having independently discovered the vagal lobes of *Heterotis*. I have seen it cited only once [W. Harder, *Anatomy of Fishes*, part I, *Text* (Schweizerbart'sche, Stuttgart, 1975), p. 377.

8. Specimens were anesthetized with MS-222. Horseradish peroxidase (Boehringer-Mannheim), on the tip of a minuten insect pin, was applied to a small localized region of an epibranchial organ. After surviving 4 to 10 days, the animals were killed and their brains and epibranchial organs removed and processed to demonstrate the distribution of HRP with either tetramethylbenzidine or Hanker-Yates reagent as the chromogen [M.-M. Mesulam, *J. Histochem. Cytochem.* **26**, 106 (1978); J. S. Hanker, P. E. Yates, C. B. Metz, A. Rustioni, *Histochem. J.* **9**, 789 (1977)].
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12. I thank R. P. Vari for aid in obtaining preserved specimens of *H. niloticus* from the fish collection of the National Museum of Natural History; G. Meola for aid in obtaining live specimens of *Heterotis*; J. P. O'Reilly and A. Kinkler for histological assistance; G. E. Meredith for translating portions of d'Aubenton's paper from French into English; O. Rodbell for photographic assistance; W. M. Saidel for advice on photography, loan of photographic equipment, helpful discussions, and comments on the manuscript; C. A. McCormick for helpful discussions and comments on the manuscript; and T. E. Finger for comments on the manuscript. Some of the material in this report was presented at the 1982 meeting of the J. B. Johnston Club. Supported by NSF research grant BNS 18844.

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Molecular Analysis of the t(2;14) Translocation of Childhood Chronic Lymphocytic Leukemia

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Two rare cases of chronic lymphocytic leukemia (CLL) in children have been studied; both are associated with a previously undescribed chromosomal translocation [t(2;14)(p13;q32)]. In one patient the translocation was reciprocal and the breakpoint on chromosome 14 occurred just 5′ of the $C_{\gamma 2}$ region on the productive immunoglobulin heavy-chain allele. The breakpoint on chromosome 2 does not involve the κ locus but lies within an uncharacterized region that coincides with the position of a constitutive fragile site that occurs within normal lymphocytes. Data on the second patient are consistent with these findings and suggest that these cases represent a rare but distinct subgroup of CLL's with a specific cytogenetic change.

MALIGNANCIES ARE OFTEN ASSOciated with specific chromosomal translocations which are now thought to represent a discrete step in tumor development (*1*). Chronic lymphocytic leukemia (CLL) is the most prevalent form of leukemia in Western countries, but cytogenetic abnormalities associated with the disease are variable (*2*). Approximately half of all CLL's exhibit a normal karyotype. The most frequent cytogenetic change observed is trisomy 12. Translocations involving 14q32, which are common in Burkitt's and follicular lymphomas (*3*), are found in only 10 percent of CLL tumors.

CLL is found almost exclusively in the middle-aged to elderly adult population (*4*). However, two children (L.H. and A.S.) were recently diagnosed as having a B-cell leukemia closely resembling CLL (*5*). The tumor cells of both patients display a previously undescribed chromosomal translocation with breakpoints occurring in band 13 on the short arm of chromosome 2 and band 32 on the long arm of chromosome 14 [t(2;14)(p13;q32)] (Fig. 1).

The proximity of the t(2;14) breakpoints to the mapped position of the immunoglobulin heavy chain (IgH) and immunoglobulin κ chain (Igκ) loci (*6*), and the involvement of these genes in the translocations of other B-cell malignancies, suggested that Ig genes or their associated sequences may have played a role in the generation of the t(2;14) translocation.

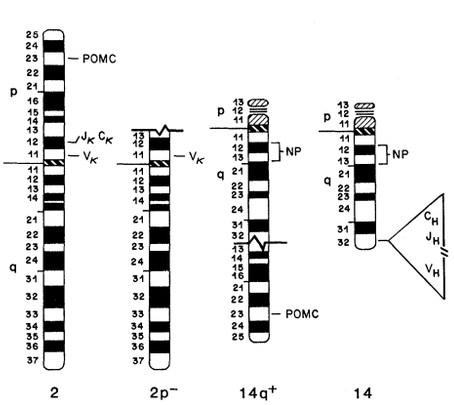

Fig. 1. Tumor chromosomes 2, 2p−, 14q+, and 14. A diagramatic representation of the translocated chromosomes is compared with that of the normal alleles. The breakpoints for both patients were found to be at 2p13 and 14q32 (*5*). The p and q denote the short and long arm, respectively, for each chromosome. Chromosomal structure is as presented in ISCN (*10*). The location of the genes for POMC, NP (*11*), and the immunoglobulin heavy- and light-chain gene segments are shown (*6*). The added material on the 14q+ is derived from the short arm of chromosome 2 (leaving a 2p−), but it could not be determined by cytogenetic analysis whether or not the translocation was reciprocal (that is, whether or not a small portion of chromosome 14 resides on the 2p−). Fibroblasts from patient L.H. displayed a normal karyotype and could not be induced to express any constitutional fragile sites (*12*).

The specific involvement of Ig sequences was determined by isolating the tumor chromosomes of interest from patient L.H. within separate somatic cell hybrids. These cell lines were then analyzed by several methods for the presence of specific genes (Fig. 1 and Table 1). Heterohybrid 4-D2 had the untranslocated tumor chromosome 14. It retains one of two tumor Ig joining region (J_H) alleles (Fig. 2A), $C_{\alpha 1}$ and $C_{\alpha 2}$ (Fig. 2B), as well as the more centromeric marker for chromosome 14, nucleoside phosphorylase (NP), but lacks any of the genes located on chromosome 2 (Table 1). The hybrid 8-D5.IB5 has the other J_H allele, as well as C_μ (Fig. 2A), C_δ (Table 1), and $C_{\alpha 1}$ (Fig. 2B), but does not have the more centromeric $C_{\alpha 2}$ segment or the gene for NP (Table 1). In addition, this hybrid bears one of the tumor κ alleles but lacks the POMC gene (pro-opiomelanocortin) from the distal end of the short arm of chromosome 2. Therefore, 8-D5.IB5 has retained the tumor 2p− chromosome. The complementary set of loci corresponding to the tumor 14q+ chromosome are found within the heterohybrid 12-B3 (Table 1). These results establish that the breakpoint on chromosome 14 has occurred between $C_{\alpha 1}$ and $C_{\alpha 2}$. They also show that the translocation was reciprocal, since the more distal IgH genes of one allele are located on the tumor 2p− chromosome. The presence of a single rearranged C_μ allele in LH, and the absence of C_μ and C_δ in 4-D2, reveals that the untranslocated tumor chromosome 14 has undergone a recombination event within the IgH locus resulting in the loss of these gene segments. The tumor cells of patient A.S. also had a single rearranged C_μ gene segment (Fig. 2A), suggesting that a similar event had occurred in

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these tumor cells as well. The size reductions observed for the C_μ bearing Hind III fragment in both tumor cell populations probably result from deletions within the switch region (S_μ).

Immunoglobulin M (IgM) is expressed on the surface of LH tumor cells, yet the translocation has left the only C_μ gene segment on the 2p− chromosome. Both μ and κ chains were detected in culture supernatants of heterohybrid 8-D5 which has the tumor 2 and 2p− chromosomes (Table 1 and Fig. 2C). Therefore, the translocation has occurred on the productive heavy-chain allele, resulting in μ production from the 2p− chromosome.

The breakpoint within the IgH locus was determined by means of the strategy shown in Fig. 3A. The 14q+ heterohybrid 12-B3 contains both the Hind III fragments downstream of the $C_{\gamma 2}$ switch region ($S_{\gamma 2}$) (K and L) (Fig. 3C). We have also detected $C_{\gamma 2}$ and $C_{\gamma 4}$ within this heterohybrid, using a constant region probe. The fragments 5′ to $S_{\gamma 2}$ (including H), as well as a 3.8-kilobase (kb) rearranged fragment also present in tumor DNA, were found in the 2p− heterohybrid (8-D5.IB5). Therefore the breakpoint occurred within the Hind III fragment, which contains the $S_{\gamma 2}$ sequences (Fig. 3B, band I). The nonproductive IgH allele in 4D-2 has the germline Hind III fragments 5′ of $C_{\gamma 2}$ and $C_{\gamma 4}$ (bands H, I, K, and L; Fig. 3C) but has lost the germline $C_{\gamma 3}$ and $C_{\gamma 1}$ fragments (bands B, E, and F). A longer exposure (Fig. 3D) reveals that this untranslocated allele has a fainter rearranged fragment of 3.0 kb which is also present in tumor DNA. Thus, the unexpressed IgH allele has undergone a switch recombination to $C_{\gamma 1}$. A diagrammatic representation of both LH tumor IgH alleles is shown in Fig. 4. The J_κ and C_κ gene segments were not interrupted by the t(2;14) in either tumor cell population (*7*), and these sequences were located proximal to the breakpoint on the 2p− chromosome iso-

Table 1. Summary of data on LH tumor cells and heterohybrids. Peripheral blood mononuclear cells from patient LH were fused with the mouse myeloma line SP 2/0 essentially as described by Fazekas *et al.* (*13*). Resulting heterohybrids as well as the fusion parents were analyzed by Southern blotting (*14*), radioimmunoassay (RIA), and isoenzyme assay for nucleoside phosphorylase (NP) (*15*). Presence or absence of the human gene (or protein in the RIA) is denoted in the data by the symbol + or −, respectively; P denotes the productive allele; N denotes the nonproductive allele; ND, not determined. The tumor chromosomes retained by individual heterohybrids are denoted in parentheses.

Cells and hybrids	Allele					Isoenzyme assay	RIA				
	J_H	C_μ	C_δ	$C_{\alpha 1}$	$C_{\alpha 2}$	NP	IgM	κ	J_k	C_k	POMC
SP 2/0	−	−	−	−	−	−	−	−	−	−	−
LH	P	+	+	+	+	ND	+	+	+	+	+
	N	−	−	+	+				+	+	
4-D2 (14)	N	−	−	+	+	+	−	−	−	ND	−
12-B3 (14q+)	−	−	−	−	+	+	−	−	−	ND	+
8-D5.IB5 (2p−)	P	+	+	+	−	−	ND	−	+	+	−
8-D5 (2,2p−)	P	+	ND	ND	ND	−	+	+	+	+	ND
									+	+	

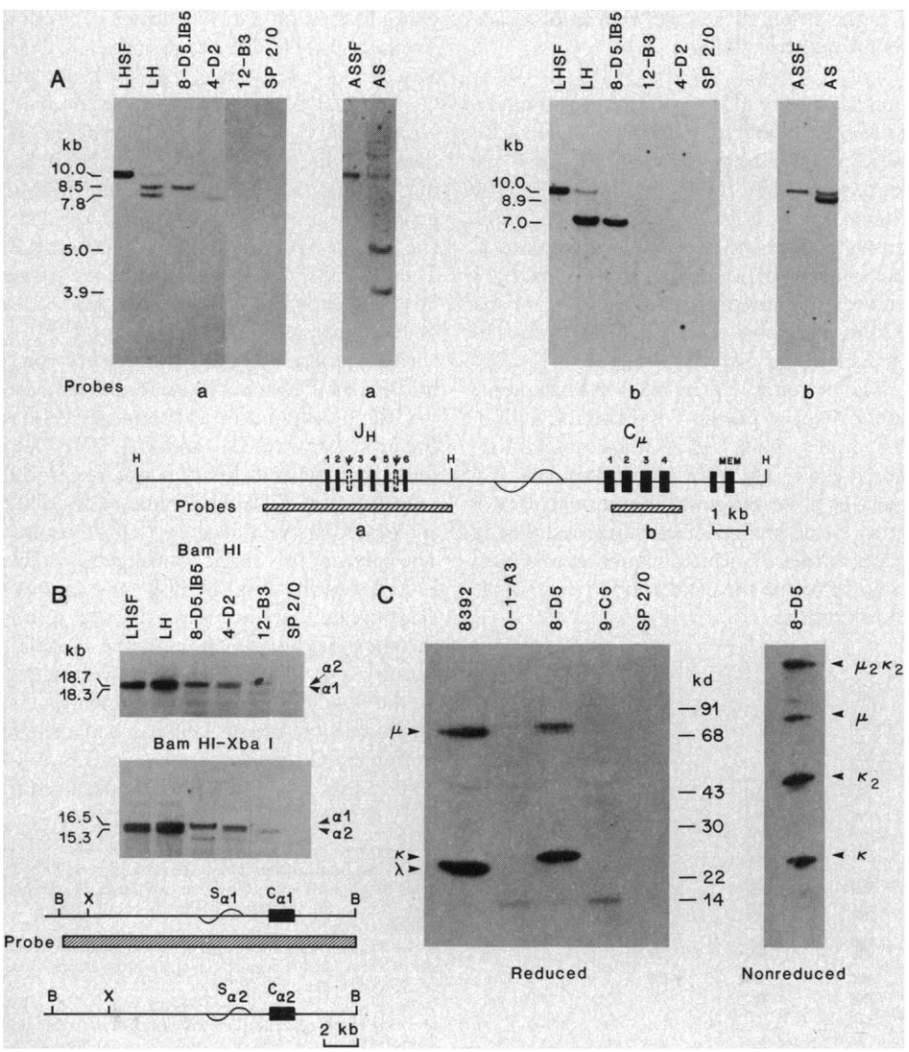

Fig. 2. Southern blot analysis of tumor and heterohybrid DNA's. LHSF and ASSF denote cultured skin fibroblast DNA from either patient. LH and AS designate DNA from peripheral blood mononuclear cells obtained from either patient during a leukemic phase of the disease. (A) All DNA's were digested with the restriction endonuclease Hind III. The J_H (a) and C_μ (b) probes utilized are designated by hatched boxes beneath the map (*16*). In both tumor DNA's normal cells contaminating the PBL (peripheral blood lymphocytes) fraction contribute a weaker hybridizing germline signal in both J_H and C_μ blots. (B) DNA's were digested and probed with a plasmid that contains the constant and switch region sequences of the human germline α_1 gene (*17*). This probe cross-hybridizes with the human α_2 locus as well. Both $C_{\alpha 1}$ and $C_{\alpha 2}$ germline maps are derived from Hisajima *et al.* (*18*). Filters in both panels were washed at 65°C, 0.1× standard saline citrate (SSC) for 2 hours. (C) The results of immunoprecipitation and sodium dodecyl sulfate–polyacrylamide gel electrophoresis analysis of culture supernatants from metabolically labeled cells (*19*). The antiserum used was an affinity-purified polyspecific rabbit antiserum specific for human Ig (Cappel). RPMI 8392 is a human B lymphoblastoid cell line which secretes both μ and λ chains. SP 2/0 was the cell line used as the fusion parent; 0-1-A3, 8-D5, and 9-C5 are individual heterohybrids. The parental line 8-D5 was used because it produces κ (from chromosome 2) which stabilizes the expression of μ. The positions of the molecular weights for the reduced side of the gel are shown.

lated from patient L.H. (Table 1). Therefore, the translocation has occurred some distance telomeric to the κ locus.

The AS tumor cells also have two S_γ rearranged bands: one hybridizes strongly (3.5 kb), and the other weakly (7.5 kb) (Fig. 3, C and D). As in LH tumor DNA, the weakly hybridizing fragment probably results from an isotype recombination. Such an event was predicted by the loss of C_μ from the unexpressed IgH allele (Fig. 2A). In addition, the $S_{\gamma 3}$- and $S_{\gamma 2}$-containing fragments (Fig. 3C, bands B and I) are reduced in intensity, which suggests that the AS rearrangements have occurred within these regions. The presence of both the fragments 5′ of $C_{\gamma 1}$ in full germline complement (Fig. 4C, bands E and F) indicates that the switch recombination in AS tumor cells has occurred within the $S_{\gamma 3}$ region. It thus seems that the stronger hybridizing rearranged fragment of 3.5 kb is due to the translocation and, further, that the breakpoint has occurred within the same $S_{\gamma 2}$-bearing Hind III fragment as observed in the LH translocation.

We suggest that these cases represent a rarely occurring but distinct subgroup of CLL's with a specific cytogenetic change that may be characteristic of an unusual pathway to the development of this particular malignancy. This translocation may provide a growth advantage for B cells specifically during the pubertal developmental period. The finding that the translocation in LH tumor cells has taken place on the productively rearranged allele was unexpected. Other translocations involving IgH genes studied previously have occurred on the nonproductive allele. This may reflect an alternative mechanism of translocation, or it may signal a selective growth advantage conferred by the involvement of the expressed allele.

Involvement of S_γ sequences in both tumor translocations suggests that the recombinases that normally effect isotype switching were responsible for generating the t(2;14). The occurrence of such recombinations on the nonproductive allele in both tumors indicates that the tumor progenitor cells were at a similar stage of differentiation, perhaps attempting an isotype switch on the expressed allele at the time that a clonal population was established. Deletions within S_μ in both tumors suggest the possibility that IgH genes on the expressed allele were in a recombinationally active state within the tumor progenitor B cell.

The translocation brings a region of chromosome 2 into the position on chromosome 14 normally occupied by a V_H region (variable region of the heavy-chain gene) after an isotype switch recombination (Fig. 4). Rearranged V_H regions can undergo extensive somatic mutations in γ-producing B cells; thus the t(2;14) may have resulted in the somatic mutation of an as yet undiscovered gene on chromosome 2. The only analogous situation reported is that of the Raji Burkitt's lymphoma cell line, which has a translocated c-*myc* gene 5′ of γ1 (*8*). In contrast to the *myc* translocations occurring near J_H and C_μ, the *myc* gene in this instance was littered with mutations. Alternatively, since $C_{\gamma 2}$ is the most 5′ member of the second C_H duplication unit and has not yet been linked to the upstream cluster (*6*), sequences that are disrupted by the translocation and that contribute to tumor development may exist upstream of $C_{\gamma 2}$.

The specific involvement of the IgH locus

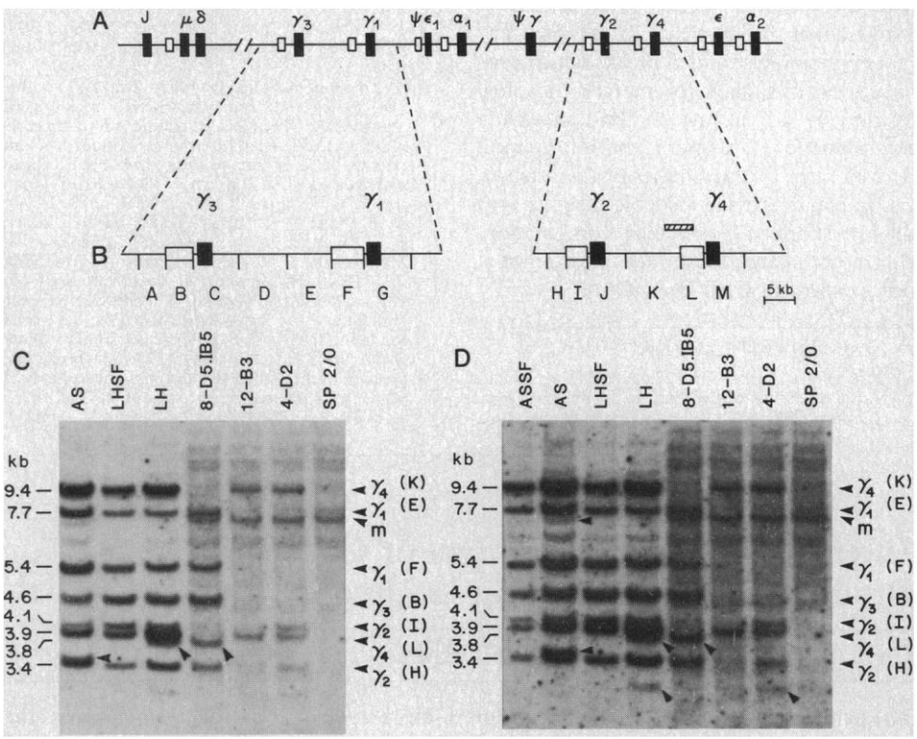

Fig. 3. Mapping the breakpoint. (A) Diagramatic representation of the overall organization of the human IgH locus (*6*). Closed boxes represent constant regions and open boxes denote switch regions in both (A) and (B). A map of the area surrounding the four functional C_γ genes (*20*) is shown to scale in (B). Hind III sites are designated by vertical lines under the map, and the various fragments generated are denoted A–M. The hatched box designates the probe used in (C) and (D). Fragment A is either not recognized or comigrates with fragment B. (C and D) 18- and 78-hour exposures, respectively, of a Southern blot in which DNA's were digested with Hind III. Fragments are identified to the right of the figure by the constant region gene they precede, and the letter in parentheses designates their position in the germline map in (B). Arrows indicate rearranged fragments. The ~6-kb fragment visible in the 78-hour exposure is present in all DNA's. This filter was washed at 68°C, 0.1× SSC for 2 hours.

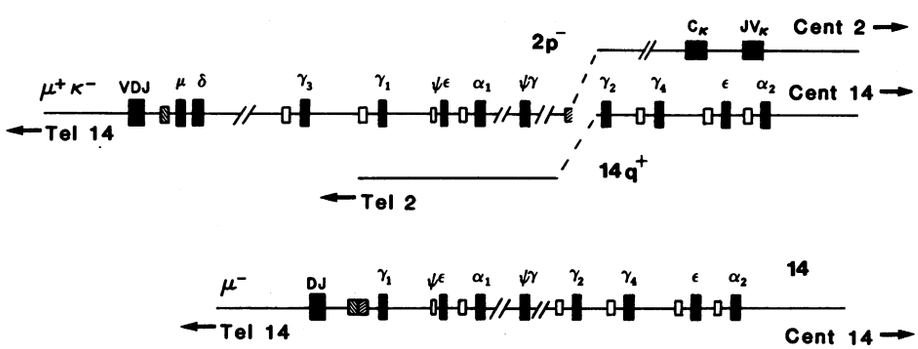

Fig. 4. LH tumor IgH alleles. A map of the two IgH alleles is shown with respect to the telomere (Tel) and centromere (Cent) of chromosomes 2 and 14. μ^+ denotes the allele that produces μ, and μ^- indicates the nonproductive IgH allele; we have not determined whether this allele has rearranged a V_H gene segment. Likewise κ^- designates the nonproductive κ allele. Constant and switch regions are represented by closed and open boxes, respectively. Switch regions that have undergone some form of rearrangement are hatched.

in the t(2;14) provides a means for studying a previously uncharacterized region of chromosome 2. Although band 2p13 has not been implicated in other translocations, nor is it a constitutional (inherited) fragile site, Yunis and Soreng report that this area is a constitutive fragile site in normal human lymphoid cells (*9*). They found that the position of other such sites correlates with the cytogenetic location of proto-oncogenes and chromosomal breakpoints associated with human malignancies. These sites may represent single-stranded breaks which provide free ends suitable for interchromosomal ligation by an enzyme involved in switch recombination. Therefore, the information derived from further studies of this translocation could provide valuable insights into the interrelations of somatic recombination, chromosomal translocation, and the unusual development of CLL in children.

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Interspecific Genetic Control of Courtship Song Production and Reception in *Drosophila*

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The genetic control of courtship song differences between *Drosophila melanogaster* and *Drosophila simulans* males was investigated by producing hybrids from reciprocal crosses. The song rhythm difference between the parental species appears to be due to sex-linked genes, whereas the basic interpulse-interval difference is autosomally inherited. Hybrid females show selective preferences for artificially generated songs carrying intermediate "hybrid" characteristics.

THE STUDY OF ACOUSTIC COMMUNICATION in hybrids has provided insight into the genetic mechanisms controlling this social interaction. In crickets, certain features of the male's calling song are autosomally inherited, whereas others are sex-linked (*1*). In both tree frogs and crickets, hybrid females respond more favorably to the songs of hybrid males than to the parentals (*2*). The possibility that the same set of genes influences both the male transmission and the female reception of song has long been discussed (*3*).

We have genetically analyzed the male lovesong in hybrids between the sibling species *Drosophila melanogaster* and *Drosophila simulans*. The song, which is produced by the male's wing vibration, stimulates the female to mate. A major component of this signal is a series of pulses with interpulse intervals (IPI's) that are typically 30 to 40 msec in *D. melanogaster* and 45 to 55 msec in *D. simulans* (*4*). The IPI's fluctuate rhythmically as the male sings, with the former species having an IPI cycle of approximately 50 to 60 seconds and the latter a 30- to 40-second cycle (*5*). We have produced two types of hybrid males between these two species, both of which have an autosome from each parent, but whose X chromosome is derived solely from one or other parental species. The well-known unisexuality of progeny produced by reciprocal crosses between the two species (*6*) usually makes it impossible to produce male hybrids with *D. melanogaster* X chromosomes. We overcame this difficulty by using a mutation in *D. simulans* which gives viable male hybrids carrying the *D. melanogaster* X chromosome. To produce a hybrid male with a *D. simulans* X chromosome, we crossed *D. melanogaster* females carrying attached-X chromosomes to males from our *D. simulans* strain. The reciprocal male carrying the *D. melanogaster* X chromosome was generated by crossing *melanogaster* females homozygous for the *yellow* body color mutation, to *D. simulans* males carrying the autosomal *Lhr* mutation which rescues the otherwise lethal male genotype from such a *melanogaster/simulans* pairing (*7*). Males from the *D. simulans Lhr* stock were also mated to *D. melanogaster* females homozygous for the *per* mutations, per^S, per^1, and per^o which, respectively, shorten, lengthen, and obliterate both circadian and song cycles (*5, 8*).

Figure 1 illustrates some of the song profiles produced by parental and hybrid males and Table 1 summarizes the data. Males from the *yellow D. melanogaster* strain show the typical *D. melanogaster* song pattern with an IPI value of 34 msec and a song oscillation period of 58 seconds. Our *D. simulans* males sang with characteristically higher IPI's of 49 msec and the shorter song cycle of 33 seconds. Compared with this *D. simulans* line, the *D. simulans Lhr* males gave a much higher IPI of 79 msec ($P < 0.01$) and a slightly longer song cycle of 39 seconds ($P < 0.05$). The large variation in the IPI among *D. simulans* strains has been documented (*9*). The male hybrid carrying

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