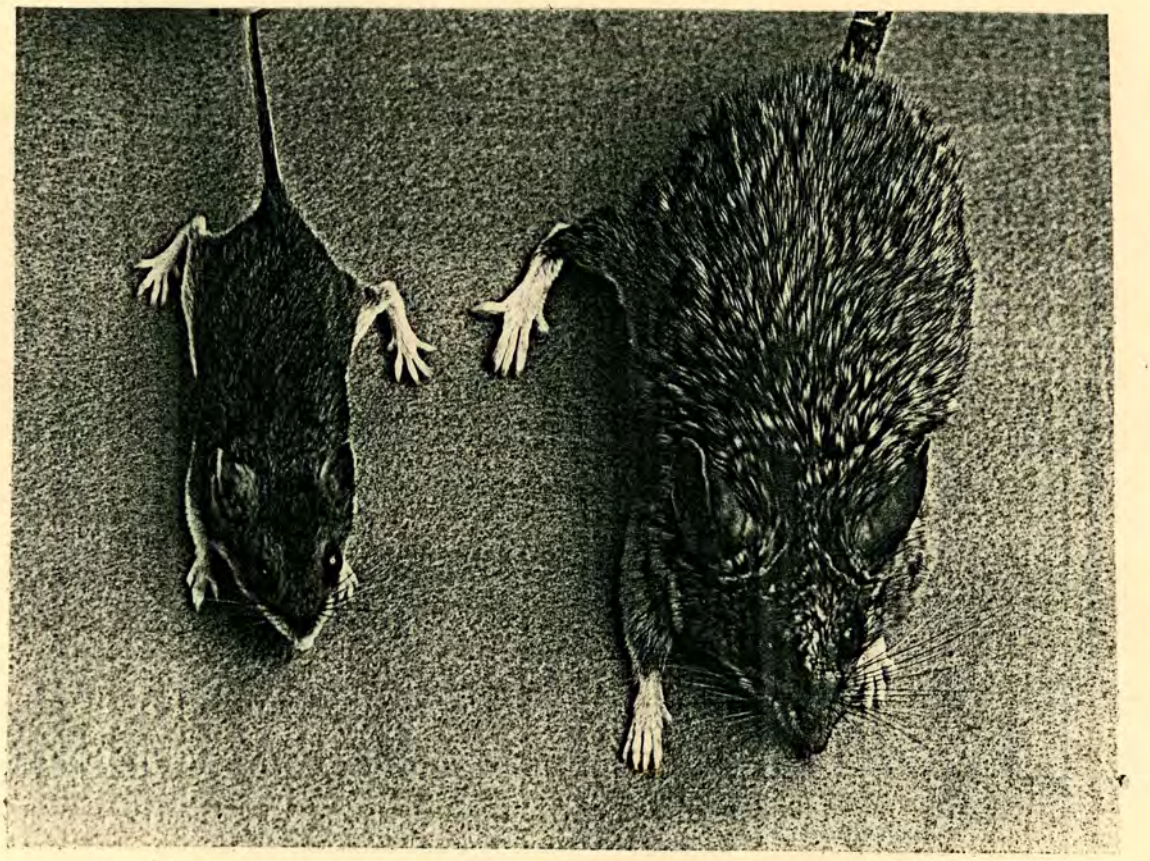
PEROMYSCUS NEWSLETTER

NUMBER SEVEN



MARCH 1989

Cover: *Peromyscus polionotus lucubrans* and *P. californicus californicus*, respectively the smallest and largest species of the genus in the United States. (15% reduction. Photo by Clint Cook)

In this Issue .....

This is the seventh issue of PEROMYSCUS NEWSLETTER. The March issue, as usual, emphasizes basic information on genetics of the deer mouse (*P. maniculatus*), including updates of gene lists and the linkage map. Similar information about other species will be given in the September 1989 issue. Any readers aware of published information which was omitted should please call it to our attention for correction in the future.

Elizabeth Barto is our featured "Peromyscus Pioneer" for this issue. Elizabeth Horner of Smith College and Suellen Van Ooteghem graciously authored the biographical sketch. Dr. Horner was associated with Dr. Barto during the 1940's when both were working at the University of Michigan with Lee R. Dice. Dr. Van Ooteghem was a laboratory assistant for Dr. Barto during the early 1970's. Dr. Barto was the "keeper of the flame" for *Peromyscus* genetics for many years, bridging the perior between R. R. Huestis and Dice and the era of protein electrophoresis. Her paper with Charles Shaw describing inheritance of lactate dehydrogenase subunits was the first demonstration of the genetics of an allozymic character in the deer mouse. One has only to scan the literature citations given in the tables in this issue of PN to appreciate the impact Betty Barto had on *Peromyscus* genetics.

We need your contributed entries. The number of entries for PN is declining, in spite of an increasing number of requests to be added to the mailing list. Please consider letting us know what is happening with your Peromyscus research. Continuation of PN depends upon the "news" from the research field!

PEROMYSCUS NEWSLETTER will publish entries up to two single spaced pages in length. PN is not a formal publication, and **entries are not to be cited without permission of the contributor.** We welcome accounts of on-going research, tentative results or information which contributes to "networking" among researchers using *Peromyscus* and other peromyscine rodents. Notices and announcements suitable for the "News and Comment" section are also invited. No charge.

We hope you find Issue #7 useful. Corrections and suggestions for changes are welcome.

W. D. D.

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NEWS AND COMMENT

Back Issues of PEROMYSCUS NEWSLETTER. We have received several requests for back issues of PN. Unfortunately, our supply of most issues is depleted since we do not print many more than are required to meet our regular mailing list. At this time we still have a few back copies of Numbers 4 and 6, which we will send without charge until the stock is exhausted. From now on we will supply xeroxed copies of any other back issues at $ 4 each upon request. The charge will offset the cost of copying. Please send your request and check made to Peromyscus Stock Center.

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David Gubernick has moved to the Department of Psychology, University of Wisconsin. His colony of Peromyscus californicus is now alive and well in Madison and was not disbanded contrary to what was indicated in PN #6. We regret this misunderstanding. Dr. Dale Sengelaub retains a small P. californicus colony at Indiana University.

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Patrick Kelly, Museum of Vertebrate Zoology, U.C.-Berkeley, writes that a high incidence of an atypical coat color occurs in deer mice on Alcatraz Island in San Francisco Bay. **Becki Thompson**, a Biology honors student at the university initiated a live-trapping study on the island and found what she termed "buffy" deer mice. These animals have an overall pale brown pelage, particularly evident in dorsal tail pimentation. The phenotype of these *Peromyscus* seems to be very similar and perhaps identical to the "brown" mutant. Recessive brown mutant *Peromyscus* have been reported on several occasions. The "blond" mutant is also similar.

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Sylvain Poirier *maintains a small stock of 6 - 7 pairs of* P. maniculatus *at the Macdonald campus of McGill University.* *See page 28.*

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The long-awaited volume **ADVANCES IN THE STUDY OF *PEROMYSCUS* (RODENTIA)** edited by Gordon Kirkland and James Layne is scheduled for release late in June. This volume is based on papers presented at the symposium on *Peromyscus* at the 1985 International Theriological Congress in Edmonton. The volume is expected to be priced at $ 22 in paperback and $ 34 cloth bound. Investigators using *Peromyscus* are looking forward to this book.

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A note from Jack King.....who has been visiting around the country, attending Elderhostels and meeting people with diverse backgrounds. It is always good to hear from him. Jack promises some information for a coming issue of PN.

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We also received a complimentary note from **Emmet T. Hooper.** Dr. Hooper was the featured "Peromyscus Pioneer" in the September '88 PEROMYSCUS NEWSLETTER.

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Bob Rose informs us that **Dale Kaukeinen,** a former student of Bill Jackson at Bowling Green, is dismantling a research lab this spring and summer. He has some lab stocks, including some of *Peromyscus*, that he is interested in finding homes for. He has about 6 different stocks, including *P. maniculatus* that he brought with him from Ohio, Malaysian rats, rice and cotton rats, and some resistant strains. He works for ICI Americas conducting efficacy testing and product development of rodenticides, hence the range of species. His address is Mr. Dale Kaukeinen, Biological Research Center, P. O. Box 208, Goldsboro, NC 27530. His phone number is (919)731-5200.

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STOCK CENTER UTILIZATION. The Peromyscus Stock Center continues to provide live *Peromyscus* as well as preserved animals and tissues to researchers. Most of the requests are for live animals to be used in various anatomical, physiological and behavioral projects. *P. maniculatus* and *P. leucopus* wild-types are the most requested. Deer mouse coat color and ADH variants have also been supplied to multiple users. Requests have been filled from institutions and indivduals in sixteen states, as well as West Germany and Japan. The Stock Center will begin providing DNA from the available stocks, and also expects to soon have Peromyscus DNA libraries and a few gene clones available.

The next few pages have additional information about stocks maintained in the center.

External Utilization of the Stock Center[1]

Year	Number of requests	Live animals	Preserved animals	Tissues[2]	DNA	Total specimens supplied
1986	10	170	0	8[3]	0	178[3]
1987	7	466	0	0	0	466
1988	8	146	312	0	0	458
1989 (to date)	8	113	32	6	0	151
Totals	33	895	344	14	0	1253

[1] Does not include animals supplied for contract or other externally funded research at the University of South Carolina.

[2] Includes skins.

[3] Does not include blood samples.

PEROMYSCUS STOCK CENTER

What is the Stock Center? The deer mouse colony at the University of South Carolina has been designated a genetic stock center under a grant from the Biological Resources Program of the National Science Foundation. The major function of the Stock Center is to provide genetically characterized types of **Peromyscus** in limited quantities to scientific investigators. Continuation of the center is dependent upon significant external utilization, therefore potential **users are encouraged to take advantage of this resource.** Sufficient animals of the mutant types generally can be provided to initiate a breeding stock. Somewhat larger numbers, up to about 50 animals, can be provided from the wild-type stocks.

A user fee of **$5 per animal** is charged and the user assumes the cost of air shipment. Animals lost in transit are replaced without charge. Tissues, blood, skins, etc. can also be supplied at a modest charge. Write or call for details.

Stocks Available in the Peromyscus Stock Center:

WILD TYPES	ORIGIN
P. maniculatus bairdii (BW Stock)	Closed colony bred in captivity since 1948. Descended from 40 ancestors wild-caught near Ann Arbor MI
P. polionotus subgriseus (PO Stock)	Closed colony since 1952. Derived from 21 ancestors wild-caught in Ocala Nat'l. Forest FL. High inbreeding coefficient.
P. leucopus (LL Stock)	Derived from 38 wild ancestors captured between 1982 and 85 near Linville NC. Third to fifth generations in captivity.
P. maniculatus X *P. polionotus* F_1 Hybrids	Sometimes available.

MUTATIONS AVAILABLE FROM THE STOCK CENTER

Coat Colors	**ORIGINAL SOURCE**
Albino *c/c*	Sumner's albino deer mice (Sumner, 1922)
Black (Non-agouti) *a/a*	Horner's black mutant (Horner *et al.*, 1980)
Blonde *bl/bl*	Mich. State colony (Pratt and Robbins, 1982)
Brown *b/b*	Huestis stocks (Huestis and Barto, 1934)
Dominant spotting *S/-*	Wild caught in Illinois (Feldman, 1936)
Gray *g/g*	Natural polymorphism. From Dice stocks (Dice, 1933)
Ivory *i/i*	Wild caught in Oregon. (Huestis, 1938)
Pink-eyed dilution *p/p*	Sumner's "pallid" deer mice. (Sumner, 1917)
Platinum *pt/pt*	Barto stock at U. Mich. (Dodson *et al.*, 1987)
Silver *si/si*	Huestis stock. (Huestis and Barto, 1934)
White-belly non-agouti a^{W}/a^{W}	Egoscue's "non-agouti" (Egoscue, 1971)
Wide-band agouti $A^{Nb}/-$	Natural polymorphism. Univ. Michigan stock (McIntosh, 1954)
Yellow *y/y*	Sumner's original mutant. (Sumner, 1917)

Note: Some of the coat color mutations are immediately available only in combination with others. For example, silver and brown are maintained as a single "silver-brown" double recessive stock. Write the Stock Center or call (803) 777-3107 for details.

Other Mutations and Variants	ORIGIN
Alcohol dehydrogenase negative *Adh^{o}/Adh^{o}*	South Carolina BW stock. (Felder, 1975)
Alcohol dehydrogenase positive *Adh^{f}/Adh^{f}*	South Carolina BW stock. (Felder, 1975)
Epilepsy *ep/ep*	U. Michigan *artemisiae* stock. (Dice, 1935)
Flexed-tail* *f/f*	Probably derived from Huestis flexed-tail (Huestis and Barto, 1936)
Hairless-2 *hre/hre***	Egoscue's hairless (Egoscue, 1962)
Juvenile ataxia *ja/ja*	U. Michigan stock. (VanOoteghem, 1983)

Enzyme variants. Wild type stocks given above provide a reservoir for several enzyme and other protein variants. See Dawson, *et al.* (1983).

*Available only on pink-eye dilution background.
**Temporarily unavailable.

Limited numbers of other stocks, species, mutants and variants are on hand, or under development, but are not currently available for distribution. For additional information or details about any of these mutants or stocks contact:

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The Advisory Committee for the Peromyscus Stock Center:

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Ira F. Greenbaum (Texas A and M University)
Clement L. Markert (North Carolina State University)
Joseph H. Nadeau (Jackson Laboratory)
Suellen Van Ooteghem (Westinghouse Corporation)
Wallace D. Dawson (University of South Carolina)

ELIZABETH BARTO

Perhaps somewhat in the same metaphorical sense that Francis B. Sumner has been called the "Father of *Peromyscus* genetics" (see **PN #2**) Elizabeth Barto could be considered the "Grand-daughter" in our academic family of pioneering peromyscan geneticists. Her introduction to the lineage was as a student of Ralph R. Huestis (**PN #3**); while he, in turn had been a student of Sumner. Like her two predecessors, Elizabeth Barto contributed importantly to early investigations of genetic markers and linkage relationships.

Elizabeth Barto was born January 29, 1908, in Thompson Falls, Montana. Her parents were William Allen Barto and Lucy Reid. William Barto, who spent his early life in Sauk Centre, some 100 miles northwest of Minneapolis, graduated from the University of Minnesota with a major in history. His wife, a normal school graduate in Minnesota, became a grade-school teacher. Following the move to Thompson Falls, William established there his own bank, a bank that remains to this day the only bank in town. Elizabeth, the third of four children, two girls and two boys, attended school in Thompson Falls and continued on at the University of Montana, in Missoula, graduating as a zoology major in 1930.

By the end of her senior year at Missoula, Elizabeth - or Betty as she is more generally known - had accepted the position of "assistant to Dr. Huestis" at the University of Oregon. It was there that she was introduced to both *Peromyscus* and the techniques of genetic analysis, working in the laboratory where new variants being studied by Huestis were augmented by mutant stocks recently received by him from Sumner. Within a short time, beginning in 1932, the Huestis-Barto collaboration produced published accounts of the inheritance of yellow, brown, silver, flexed-tail, tremor, and white star. With Huestis as her mentor as well as her employer, Betty received her M. A. in zoology in 1932 and, in 1935, departed for Michigan at the invitation of Lee R. Dice.

Her first two years in Michigan were spent in Bloomfield Hills as secretary of the Cranbrook Institute of Science, where Dice served as a trustee. Here her analytical abilities and her meticulous attention to details were acknowledged by junior authorship on one of the Institute's bulletins (No. 9. **A Pictorial Guide to the Families of Birds** [of Eastern North America]. Edward T. Boardman and Elizabeth Barto, 52 pp., 74 figures. October 1937).

In 1937 Betty became Dice's full-time secretary and assistant at the University of Michigan, serving as his secretary in his position as Curator of Mammals in the Museum of Zoology and as his assistant in his position as Director of the Laboratory of Vertebrate Genetics (**PN #4**). The following year, with appointment of Emmet T. Hooper to the Museum's Curatorship of Mammals (**PN #6**), Betty became Dice's full-time research assistant at the Laboratory of Vertebrate Genetics, which, with its gradually broadening philosophy, underwent a change of name in 1942 to the Laboratory of Vertebrate Biology, During the war years she took charge of much of the work in mammalian biology and in 1944 was promoted to junior biologist. The new title affected only minimally the nature of her responsibilities even when, in 1950, the Laboratory became an operating unit of Dice's Institute of Human Biology. In 1955 she received a Ph.D. Her thesis research, directed by Dice, was on the peromyscan behavioral mutant known as boggler (*bg*). Elizabeth C. Crosby, Clement L. Markert, David L. Nanney, and James V. Neel all served on her doctoral committee.

Following Dr. Dice's retirement, in 1957, Dr. Morris Foster assumed directorship of the Laboratory of Vertebrate Biology as an appointee of the Department of Zoology, while Dr. Barto, as Biologist in the Department of Zoology, continued to work at the same Laboratory. The major focus of the Laboratory turned towards genetics of coat color in the house mouse. Despite the several changes in titles, affiliations, and policies, Betty was able to procure sufficient space and grant support to carry on, within a laboratory now re-christened the Laboratory of Mammalian Genetics and dedicated primarily to *Mus* genetics, a forward-looking program in *Peromyscus* genetics. Indeed, it was during this period that, in collaboration with Charles R. Shaw, she conducted some of the landmark studies of genetic variation of proteins in *Peromyscus*. Their papers describing the inheritance of lactate dehydrogenase (1963. PNAS 50:211-214) and autosomal glucose-6-phosphate dehydrogenase allozymes (1965. Science. 148:1199-1100) are particularly noteworthy. Extention of these studies and of studies using similar techniques has added greatly to our understanding of protein polymorphisms and genetic diversity in relation to *Peromyscus* speciation. Betty's name is associated with some of the earliest biochemical genetics of *Peromyscus*. as well as with characterization of morphological and behavioral mutants and with investigation of linkage relationships.

An avid reader and well-informed in art, literature, and music, as well as in science, Betty was an inspirational colleague for those of us who came to know her at Michigan. Modest, reserved, and even self-effacing, she nevertheless impressed us indelibly with her quiet efficiency and gentle humor. Her dedication to science brought her membership in the University's Women's Research Club; and, as a non-member resident for many years of the house run by the women's medical sorority, Alpha Epsilon Iota, she was particularly knowledgeable on issues in current medicine. A late worker, she seemed essentially fearless, remaining long hours, alone, in what was undoubtedly one of the most isolated laboratories on campus.

Among the peromyscan behavioral investigations conducted by Dice and Barto was that of epilepsy. In that capacity she characterized a number of independent epileptic mutant stocks of *Peromyscus*. In addition, her interest in epilepsy resulted in an enterprise that led unexpectedly to Betty's becoming for many months the almost constant observer-companion of a convulsive adult airdale named Judy. The remembered story is that the dog was brought over from Ireland by a dog breeder who, learning that Judy had recurrent seizures, presented her to the Laboratory. Undeterred by the fact that the Laboratory had neither space nor staff for handling Judy, Betty adopted her until proper quarters could be constructed for housing her and her progeny. The Alpha Epsilon Iota medical sorority received formal University acknowledgement for its role in offering the dog interim tenancy and surveillance.

Upon her retirement in 1973, Betty returned to her original family home in Thompson Falls, the town in western Montana for which she has maintained a life-long affection. Even today when the air is still the smell of wood smoke from the saw mill hangs over the town. The trout streams are clear and deep; the pace of living is slow, peaceful, and conducive to the quiet pleasures of retirement. There, where her parents pioneered in building a town, Betty, in characteristic modesty, disclaims suggestion that she, too, has pioneered. Yet, pioneering has many yardsticks. Cogently, in the bibliography of David Rasmussen's chapter on genetics in **Biology of Peromyscus** (1968, J. A. King, ed.), Elizabeth Barto leads the list of cited authors and co-authors. Her own citations, alone or in collaboration with others, number thirteen. Next comes Ralph R. Huestis, with eleven; then Francis B. Sumner, with eight. As we turn the corner and enter an new era in *Peromyscus* genetics, there again, among its pioneers in biochemical studies, is Elizabeth Barto.

B.E.H. and S.V.O.

GENETIC LOCI IN THE DEER MOUSE

(*Peromyscus maniculatus*)

Tables 1A, 1B, 1C and 1D list recognized genetic loci described in *Peromyscus maniculatus* and other species of the *maniculatus*-group. This list is limited to loci for which formal genetic analysis of crosses has been conducted and appropriately reported in the published scientific literature. Additional genetic traits are known and some have been mentioned in abstracts, casual reports, newsletters, grant proposals, papers presented at meetings, etc. The latter are not included, since the descriptions and genetics generally are not complete enough to formally define the loci.

Table 2 lists presumptive variant protein loci described from natural populations of *P. maniculatus* and other members of the *maniculatus*-species group. These loci have not necessarily been formally demonstrated by mendelian crosses. Monomorphic (invariant) protein loci are not listed, but variants among potentially interbreeding species are included. For example, one allozyme may occur in one species, *e.g. P. polionotus* and another in a different species, *e.g. P. maniculatus*. Only published reports are included.

Although the genetic nomenclature of *Peromyscus* is not yet completely standardized, the conventions used for the house mouse (*Mus*) are employed wherever possible. In designating genetic loci we adopted the symbols given by the original investigator, unless there is clear homology with *Mus* loci for which standarized symbols are assigned; or in cases where the original symbols have been superceded by subsequent usage, in which case we have used the most recent revision. However, identity of locus symbols used for *Peromyscus* with those used in other taxa of mammals does not necessarily imply homology. As a case in point, *ja* in *Mus* is the "jaundiced" locus, in *Peromyscus ja* represents "juvenile ataxia". If a variant is shown to be allelic with a previously reported gene, the locus symbol is reduced to an allelic symbol. Where two authors have used the identical symbol for different loci in *Peromyscus* we have given priority to the first reported, and devised an alternate designation for the other.

References cited in the tables are available in a list of *Peromyscus* genetic literature compiled by Dr. Bruce Buttler, Biology Department, Canadian Union College, College Heights, Alberta, Canada, TOC OZO.

Table 1A

Genetic Loci Formally Described in the *Peromyscus maniculatus* Species Group.

Coat Color and Pattern Genes.

Name of locus and alleles	Symbol	Mode of inheritance[1]	Linkage group	Definitive description and analysis	Collateral descriptions, interactions and recurrences	Recombination reported
AGOUTI			III			
Wide-band agouti	A^{Wb}	dominant		McIntosh (1956a)	Blair (1947) as "buff"	Clark (1938) as "buff"
White-belly non-agouti	a^{w}	recessive		Egoscue (1971)		
Non-agouti (Black)	*a*	recessive		Horner *et al.* (1980)		
BROWN	*b*	recessive	II	Huestis and Barto (1934)	Blair (1947), McIntosh (1956a); Dawson *et al.* (1969)	Huestis and Barto (1934), Blair (1947), Barto (1955, 1956), McIntosh (1956a)
Orange-tan	b^{or}	recessive		Egoscue and Day (1958)		
BLOND	*bl*	recessive		Pratt and Robbins (1982)		
ALBINO	*c*	recessive	I	Sumner (1922)	Clark (1938)	Sumner (1922), Clark (1936, 1938), Feldman (1937), Barto (1942a), Huestis and Lindstedt (1946), Huestis (1946)
COLORLESS HAIRTIP*	*ctp*	recessive		Bowen and Dawson (1969)	Bowen (1968)	
DILUTE*	*d*	recessive	II	Dice (1933)	Clark (1938), Barto (1942a, 1956), McIntosh (1956a)	
GRAY	*g*	recessive		Dice (1933)	Clark (1938), Blair (1947), McIntosh (1956a)	Blair (1944, 1947)
IVORY	*i*	recessive		Huestis (1938)	Clark (1938) McIntosh (1956a)	Barto (1942a, 1956),
PINK-EYED DILUTION	*p*	recessive	I	Sumner (1917) as "pallid"	Clark (1938), Barto (1942b) Feldman (1937), Snyder (1980a)	Sumner (1922), Clark (1936, 1938),
PLATINUM	*pt*	recessive		Dodson *et al.* (1987)		Dodson *et al.* (1987)
RED EYE (Heterochromia)	*r*	recessive		Huestis and Willoughby (1950)		
DOMINANT SPOT (Whiteface)	*S*	dominant		Feldman (1936)	Maddock (1966)	Feldman (1937)

Table 1A. Coat Color and Pattern (Continued)

Name of locus and alleles	Symbol	Mode of inheritance[1]	Linkage group	Definitive description and analysis	Collateral descriptions, interactions and recurrences	Recombination reported
SILVER	*si*	recessive	I	Huestis and Barto (1934)		Huestis and Barto (1934), Huestis and Piestrak (1942), Huestis and Lindstedt (1946), Barto (1956)
WHITE CHEEK	*Wc*	dominant		Blair (1944)	Bowen and Dawson (1977)	Blair (1944)
WHITESIDE	*wh*	recessive		McIntosh (1956b)		
YELLOW	*y*	recessive		Sumner (1917) Clark (1938) Barto (1956)	Sumner and Collins (1922), McIntosh (1956a)	Sumner (1922), Feldman (1937),
COMPLEXLY INHERITED TRAITS:						
Minor white spotting (star, splash, etc.)	*p-1, p-2*	recessive incompletely, penetrant		Feldman (1936)	Sumner (1932), Barto and Huestis (1933)	
Grizzled	"*G*"	"complex dominant"		Sumner (1928, 1932)		
Coat pattern in *P. polionotus*				Bowen and Dawson (1977)	Bowen (1968)	Bowen and Dawson (1977)
Pointed A	P_a	dominant	VII			
Pointed B	P_b	dominant	VII			
Tapered	*Tp*	dominant				
Coat pattern modifiers				Bowen and Dawson (1977)		
Squared modifier	*Rs*	incompletely dominant				
Tapered modifier	*Rt*	dominant				

[1]Autosomal unless other wise stated.

*No longer known to be in existence

Table 1B

Genetic Loci Formally Described in the *Peromyscus maniculatus* Species Group.

Integumentary, Skeletal and Pathological Variants.

Name of locus and alleles	Symbol	Mode of inheritance[1]	Linkage group	Definitive description and analysis	Collateral descriptions, interactions and recurrences	Recombination reported
CATARACT-WEBBED (Syndactyly)	*cw*	recessive		Anderson and Burns (1979)		
FLEXED TAIL	*f*	recessive	I	Huestis and Barto (1936)		Huestis and Barto (1936a), Huestis and Piestrak (1942), Huestis and Lindsiedt (1946), Huestis *et al.* (1956), Barto (1956)
HAIRLESS-1	*hr-1*	recessive		Sumner (1924)		Sumner (1924, 1932), Feldman (1937), Clark (1938), Barto (1942a, 1955, 1956), McIntosh (1956a)
HAIRLESS-2	*hr-2*	recessive		Egoscue (1962)		
NUDE*	*n*	recessive		Clark (1938)	Barto (1942a)	
SPHEROCYTOSIS (Inherited jaundice)	*sph*	recessive		Huestis and Anderson (1954)	Huestis *et al.* (1956) Motulsky *et al.* (1956)	Huestis *et al.* (1956)

Table 1C

Genetic Loci Formally Described in the *Peromyscus maniculatus* Species Group.

Behavior and Neurological Variants.

Name of locus and alleles	Symbol	Mode of inheritance[1]	Linkage group	Definitive description and analysis	Collateral descriptions, interactions and recurrences	Recombination reported
BOGGLER	*bg*	recessive		Barto (1955)	Vandermere and Barto	Barto (1955)
EPILEPSY (EP; waltzing in *artemisiae*)	*e* (ep, v_2)	recessive		Dice (1935) Chance and Yaxley (1950), Barto (1954, 1956)	Clark (1938), Watson (1939), Barto (1956)	Watson (1939)
JUVENILE ATAXIA	*ja*	recessive		VanOoteghem (1983)		
SPINNER* (Waltzing in *rhoadsi*)	*sp* (v_3)	recessive		Watson (1939)	Barto (1954)	
TREMOR*	*tr*	recessive		Huestis and Barto (1936b)		
WALTZER* (Waltzing in *bairdii*)	*v* (*w*)	recessive	III	Dice (1935)	Clark (1938), Watson (1939)	Barto (1942a, 1954, 1956), McIntosh (1956a)

[1]Autosomal unless otherwise stated.
*No longer known to be in existence

Table 1D

Genetic Loci Formally Described in the *Peromyscus maniculatus* Species Group.

Biochemical and Immunological Variants.

Name of locus	Allelic designation	Linkage group	Definitive description and formal analysis	Recombination reported
ALCOHOL DEHYDROGENASE (liver)	Adh^f Adh^s Adh^a	VI	Felder (1975), Burnett and Felder (1978a, 1978b)	Dawson *et al.* (1983)
ALBUMIN (serum)	Alb^{100} Alb^{96} Alb^{86}	VI	Brown and Welser (1968) Jensen and Rasmussen (1971)	Dawson (1982, Dawson *et al.* (1983)
AMYLASE (salivary)	$Amy\text{-}1^a$ $Amy\text{-}1^b$ $Amy\text{-}1^c$	VI	Evans *et al.* (1977)	Dawson *et al.* (1983)
ERYTHROCYTIC ANTIGEN	$Ea^A = (Pm^A)$ $Ea^B = (Pm^B)$ $Ea^C = (Pm^C)$	IV	Rasmussen (1961), Savage and Cameron (1971)	Randerson (1973)
ESTERASE (erythrocytic)	$Es\text{-}1^o$ $Es\text{-}1^a$ $Es\text{-}1^b$	IV	Randerson (1965), Van Deusen and Kaufman (1978)	Randerson (1973)
ESTERASES (tissue and serum)	*Es-2* through *Es-7* (Symbols not standardized)		Rasmussen and Jensen (1971), Dawson (1982), Gill (1976), Baccus *et al.* (1980)	Dawson (1982)
GLUTAMATE OXALOACETATE TRANSAMINASE (soluble)	$Got\text{-}1^a$ $Got\text{-}1^b$ $Got\text{-}1^c$		Gill (1976)	Dawson *et al.* (1983)
GLUCOSE-6-PHOSPHATE (AUTOSOMAL HEXOSE-6-P) DEHYDROGENASE (soluble)	$Gόpd\text{-}1^a$ $Gόpd\text{-}1^b$		Shaw and Barto (1965) Shaw (1966)	
a-GLYCEROPHOSPHATE DEHYDROGENASE (tissue)	$Gpd\text{-}1^a$ $Gpd\text{-}1^b$		Gill (1976)	
HEMOGLOBIN - ALPHA TYPE GLOBINS (Duplicated locus)	$Hba^1 = (Hb^f) = (HbI^a)$ Hba^2 $Hbc^o = (Hb^o) = (HbI^o)$ Hbc^1 $Hbc^2 = (Hb^s)$		Thompson *et al.* (1966), Rasmussen *et al.* (1968), Jensen *et al.* (1976), Maybank and Dawson (1976), Snyder (1978, 1980b)	
HEMOGLOBIN - BETA TYPE GLOBINS (Triplicated locus)	Hbb^1 Hbb^o *Hbb-b1* Hbd^1 or *Hbb-b2* Hbe^o *Hbb-b3* Hbe^1	I	Snyder (1978, 1980b), Padgett *et al.* (1987)	Snyder (1980a)
HAPTOGLOBIN (serum)	Hpt^1 Hpt^2		Rasmussen (1968) Griswold and Dawson (1971)	

Table 1D. Biochemical and Immunological Variants (Continued)

Name of locus	Allelic designation	Linkage group	Definitive description and formal analysis	Recombination reported
IMMUNOGLOBIN ($7Sγ_1$)	Ig^f Ig^s		Coe (1972)	
LACTATE DEHYDROGENASE A SUBUNIT (tissue)	$Ldh\text{-}A^a$ $Ldh\text{-}A^b$		Cattanach and Perz (1969)	
LACTATE DEHYDROGENASE B SUBUNIT (tissue)	$Ldh\text{-}B^f$ $Ldh\text{-}B^s$		Shaw and Barto (1963)	
LEUCINE AMINOPEPTIDASE (serum)	$Lap\text{-}1^a$ $Lap\text{-}1^b$	V	Dawson (1982) Dawson *et al.* (1983)	Dawson (1982),
SUPEROXIDE DISMUTASE	$Sod\text{-}1^f$ $Sod\text{-}1^p$ $Sod\text{-}1^M$		Birdsall *et al.* (1970)	
6-PHOSPHOGLUCONATE DEHYDROGENASE (tissue)	$Pgd\text{-}1^a$ $Pgd\text{-}1^b$		Gill (1976)	Dawson *et al.* (1983)
PHOSPHOGLUCOMUTASE-1 (tissue)	$Pgm\text{-}1^a$ $Pgm\text{-}1^b$		Gill (1976)	
PHOSPHOGLUCOMUTASE-4 (tissue)	$Pgm\text{-}4^a$ $Pgm\text{-}4^b$ $Pgm\text{-}4^c$		Gill (1976)	
TRANSFERRIN (serum)	$Trf^a = (Trf^f)$ Trf^b Trf^c Trf^e Trf^M	V	Rasmussen and Kochn (1966), Biggers and Dawson (1971), Griswold and Dawson (1971) Canham *et al.* (1970)	Dawson (1982) Dawson *et al.* (1983)

[1] Autosomal unless otherwise stated.

Table 2
Allozyme and Other Protein Loci Reported from Natural Populations of the *Peromyscus maniculatus* Species Group

Protein	Locus	Species[1]	Reference[2]
ALBUMIN	*Alb*	*P. maniculatus* *P. polionotus*	Rasmussen (1970) Jensen and Rasmussen (1971) Selander et al. (1971) Biggers and Dawson (1971) Loudenslager (1978) Baccus et al. (1980) Massey and Joule (19810
ALCOHOL DEHYDROGENASE	*Adh-1*	*P. maniculatus* *P. melanotis*	Avise et al. (1979) Baccus et al. (1980) Massey and Joule (1981)
AMYLASE	*Amy-1*	*P. maniculatus*	Aquadro and Patton (1980)
ESTERASE	*Es-1* *Es-2* *Es-3* *Es-4* *Es-5* *Es-6* *Es-7* *Es-8*	*P. maniculatus* *P. polionotus*	Rasmussen and Jensen (1971) Selander et al. (1971) Peck and Biggers (1975) Gill (1976) Loudenslager (1978) Foltz (1981) Aquadro and Kilpatrick (1981) Massey and Joule (1981)
GLUTAMATE OXALOACETATE TRANSAMINASE	*Got-1*	*P. maniculatus* *P. polionotus* *P. melanotis*	Selander et al. (1971) Gill (1976) Loudenslager (1978) Avise et al. (1979) Baccus et al. (1980) Aquadro and Kilpatrick (1981) Massey and Joule (1981)
GLUCOSE 6-PHOSPHATE DEHYDROGENASE	*G6pd-1* (H6pd-1)	*P. maniculatus*	Shaw and Barto (1965) Loudenslager (1978) Aquadro and Kilpatrick (1981)
GLUCOSE PHOSPHATE ISOMERASE	*Gpi-1* (Pgi-1)	*P. maniculatus* *P. polionotus* *P. melanotis*	Selander et al. (1971) Avise et al. (1979) Foltz (1981) Massey and Joule (1981)
a-GLYCEROPHOSPHATE DEHYDROGENASE	*Gpd-1*	*P. maniculatus* *P. polionotus* *P. oreas*	Selander et al. (1971) Mascarello and Shaw (1973) Gill (1976) Avise et al. (1979)
HAPTOGLOBIN	*Hpt*	*P. polionotus*	Peck and Biggers (1975)
HEMOGLOBIN	*Hba* *Hbb*	*P. maniculatus* *P. polionotus* *P. melanotis*	Thompson et al. (1966) Ahl (1968) Foreman (1968) Rasmussen et al. (1968) Rasmussen (1970) Selander et al. (1971) Snyder (1977,1980) Loudenslager (1978) Avise et al. (1979) Aquadro and Kilpatrick (1981) Massey and Joule (1981) Chappell and Snyder (1984)

(CONTINUED NEXT PAGE)

Table 2 (Continued)

Protein	Locus	Species[1]	Reference[2]
ISOCITRATE DEHYDROGENASE	*Idh-1*	*P. maniculatus* *P. oreas* *P. polionotus* *P. sejugis*	Mascarello and Shaw (1973) Baccus et al. (1980) Avise et al. (1974) Aquadro and Kilpatrick (1981) Massey and Joule (1981)
LACTATE DEHYDROGENASE	*Ldh-1* *Ldh-2*	*P. maniculatus* *P. polionotus* *P. melanotis*	Selander et al. (1971) Avise et al. (1979) Massey and Joule (1981)
MALATE DEHYDROGENASE	*Mdh-1* *Mdh-2*	*P. polionotus* *P. maniculatus*	Selander et al. (1971) Massey and Joule (1981)
PEPTIDASE	*Pep-1* (Pep-B)	*P. maniculatus* *P. melanotis*	Avise et al. (1979) Baccus et al. (1980) Massey and Joule (1981)
6-PHOSPHOGLUCONATE DEHYDROGENASE	*Pgd-1*	*P. maniculatus* *P. polionotus* *P. oreas*	Selander et al. (1971) Mascarello and Shaw (1973) Gill (1976) Avise et al. (1979) Baccus et al. (1980) Foltz (1981) Massey and Joule (1981)
PHOSPHOGLUCOMUTASE	*Pgm-1* *Pgm-2* *Pgm-3* *Pgm-4*	*P. maniculatus* *P. polionotus* *P. melanotis*	Selander et al. (1971) Mascarello and Shaw (1973) Gill (1976) Avise et al. (1979) Aquadro and Kilpatrick (1981) Massey and Joule (1981)
SORBITOL DEHYDROGENASE	*Sdh-1*	*P. maniculatus*	Baccus et al. (1980) Massey and Joule (1981)
TRANSFERRIN	*Trf*	*P. maniculatus* *P. polionotus*	Rasmussen (1970) Biggers and Dawson (1971) Selander et al. (1971) Gill (1976) Redfield (1976) Loudenslager (1978) Avise et al. (1979) Baccus et al. (1980) Foltz (1981) Massey and Joule (1981)
Miscellaneous Non-specific Proteins		*P. maniculatus*	Mascarello and Shaw (1973) Gill (1976)

[1]Species from which protein variants were obtained.

[2]Reference list is available from Dr. Bruce Buttler, Div. Natural & Physical Sciences, Canadian Union College, 400 College Heights, Alberta TOC OZO, Canada.

GENETIC LINKAGE MAP OF *PEROMYSCUS MANICULATUS*

Linkage data for the deermouse (*Peromyscus maniculatus*) collected before 1972 are summarized by Robinson (15, 16). The system of assigning linkage groups on the basis of a single marker employed during the 1940's and 50's (2,13) is no longer used. "Group IV" in the earlier system is now Group II, and old Groups "II" and "III" have been abandoned. In the interim since Robinson's review several additional linkages have been added (3, 8, 9, 17). Figure 1 represents the current status of the linkage map for the deermouse and its sibling species *P. polionotus*. Six linkage groups are now established by formal genetics and another is tentative. An additional linkage, *Es-5 - Es-6*, by homology with *Mus* will probably map to Group IV (8), and is designated IVa in Table 3.

The order of loci in Group I was reported informally by Huestis and Silliman in an unpublished communication, according to Robinson (15), and has been partially confirmed by Dodson (unpub.). Linkage of *Trf* and *Lap* is tentative (8), but is homologous with a similar linkage in *Mus*. The *Pep-2* locus is provisionally assigned to Group VI proximal to *Alb*, but has not been mapped further (9).

Positive, but not significant, lod scores suggesting possible linkage between the gene pairs *Adh - 6Pgd*, *Adh - Got-1*, *Adh - Idh*, *Alb - Pept-1*, *Alb - Sdh* and *Est-4 - Sdh*, respectively, were reported by Baccus *et al.* (1). Subsequent information indicates that *Adh* and *Got-1* are independent, as are the *Alb* and *Sdh* loci (9).

The *Hbe* locus is part of the triplicated beta globin site (*Hbb*), according to Snyder (17). Unpublished data from Snyder maps the position of the *Gpi-1* and *Hbe* loci relative to the albino (*c*) and pink-eyed dilution (*p*) loci. Silliman (unpub.) proposed that there is a duplication, *f'*, closely linked to the *f* locus.

Two significant markers on the *Peromyscus* linkage map. *d* and *v*, are now extinct in laboratory stocks of deermice. The "flexed tail" trait which occurs in a laboratory stock may not be identical by descent with the original trait used in early linkage studies, but it maps to the same location in Group I.

The chromosome number of all *Peromyscus* species is 2N = 48 (6). None of the linkage groups have been assigned to chromosomes, but partial banding homology between *Rattus* and *Peromyscus* chromosome 1 (12) suggests that Linkage Group I is probably located on Chromosome 1 in deer mouse as the homologous group is in rat (7).

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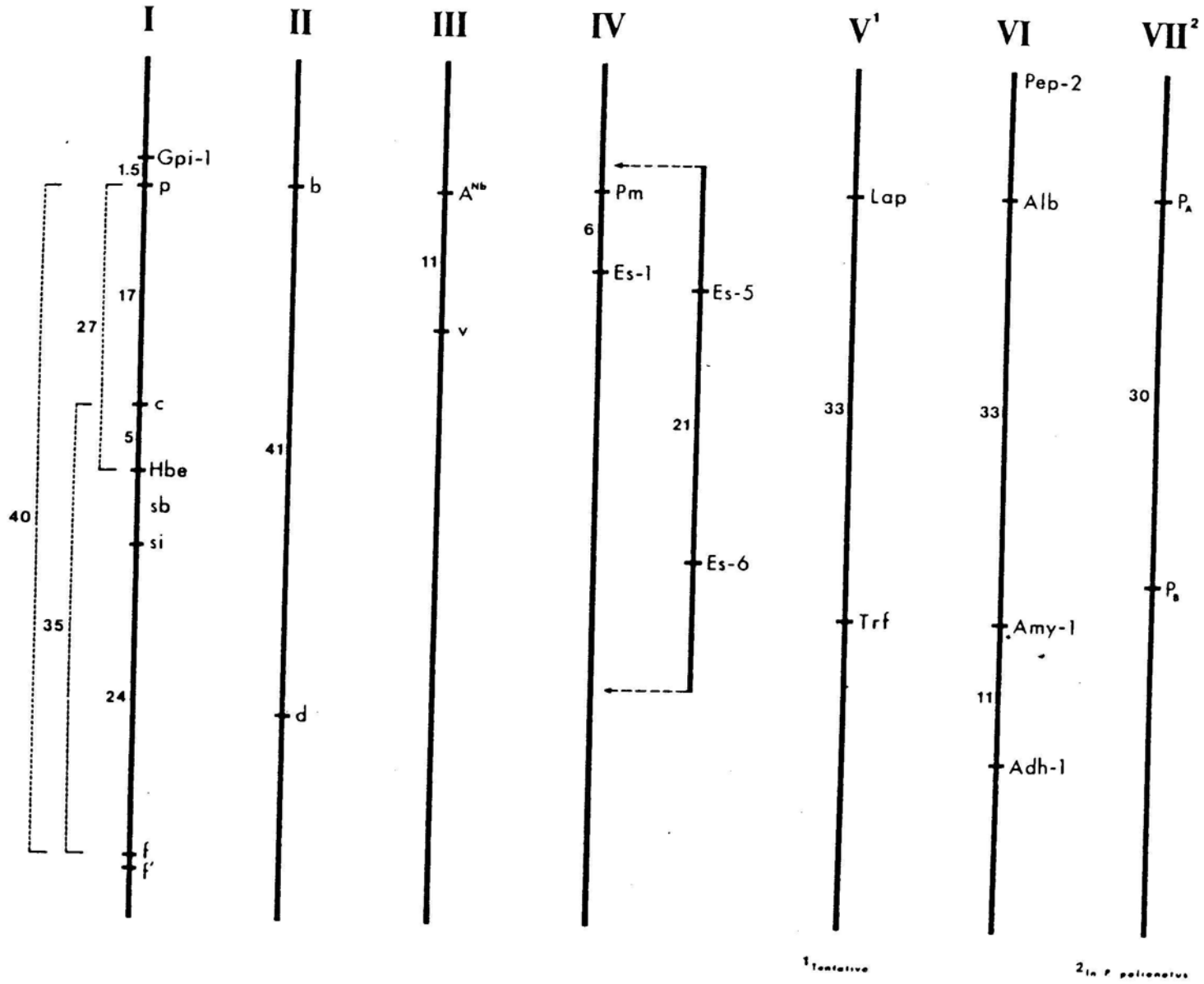
MAPPED LOCI IN *PEROMYSCUS MANICULATUS*

Gene Symbol	Name of Locus	Linkage Group	Reference
A^{Nb}	Agouti	III	McIntosh (1956)
Adh-1	Alcohol dehydrogenase (liver)	VI	Dawson *et al.* (1983)
Alb	Albumin (serum)	VI	Dawson *et al.* (1983)
Amy-1	Amylase (salivary)	VI	Dawson *et al.* (1983)
b	Brown	II	McIntosh (1956)
c	Albino	I	Sumner (1922), Clark (1936, 1938), Huestis and Lindstedt (1946)
d	Dilute	II	McIntosh (1956)
Ea	Pm erythrocytic antigen	IV	Randerson (1973)
Es-1	Esterase-1 (erythrocytic)	IV	Randerson (1973)
Es-5	Esterase-5 (kidney)	IVa	Dawson (1982)
Es-6	Esterase-6 (kidney)	IVa	Dawson (1982)
f	Flexed tail	I	Huestis and Piestrak (1942), Huestis and Lindstedt (1946)
Gpi-1	Glucose phosphate isomerase (erythrocytic)	I	Snyder (1980)
Hbb	Beta globin (hemoglobin)	I	Snyder (1980)
Lap-1	Leucine aminopeptidase (serum)	V	Dawson (1982)
p	Pink-eyed dilution	I	Sumner (1922), Clark (1936, 1938), Snyder (1980)
P_A	Pointed rump pattern A	VII	Bowen and Dawson (1977)
P_B	Pointed rump pattern B	VII	Bowen and Dawson (1977)
Pep-2	Tripeptidase (erythrocytic)	VI ?	Dawson *et al.* (1983)
sb	Snub nose	I	Robinson (1972)
si	Silver	I	Huestis and Piestrak (1942), Huestis and Lindstedt (1946)
Trf	Transferrin (serum)	V	Dawson (1982)
v	Waltzer	III	McIntosh (1956)

For reference citations see Bruce Buttler "Bibliography of *Peromyscus* (Rodentia) Genetics". 1986.

LINKAGE MAP OF THE DEERMOUSE



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N O T I C E

PEROMYSCUS NEWSLETTER IS NOT A FORMAL SCIENTIFIC PUBLICATION.

INFORMATION AND DATA IN THE "CONTRIBUTIONS" SECTION

SHOULD NOT BE CITED OR USED

WITHOUT PERMISSION OF THE CONTRIBUTOR.

THANK YOU!

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CONTRIBUTIONS

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It is known that fructose stimulates ethanol metabolism in vivo in the perfused rat liver and in ADH positive deermice by decreasing ATP, thereby increasing NADH supply for ADH; however, in perfused liver from ADH negative deermice, fructose decreased rates of ethanol metabolism by nearly 70%. Fructose also decreased H_2O_2 supply in perfused livers from the ADH^- deermouse, implicating catalase in this phenomenon (Handler et al. Biochem. J. 248:415, 1987). Therefore, the effect of fructose on the increase in ethanol metabolism due to chronic treatment with ethanol was studied in ADH^- deermice. Rates of ethanol elimination were increased by a factor of 2 to 3 by treatment for at least two weeks with modified Lieber-Decarli liquid ethanol diet. Fructose (1g/kg, i.p.) decreased rates of ethanol elimination by approximately 30% in control and 50% in ethanol-treated ADH^- deermice, suggesting strongly that the increase in ethanol elimination due to chronic ethanol treatment is due to catalase. In rodents, methanol and butanol are selective substrates for P-450 and catalase, respectively. Based on minimal metabolism of butanol, it was concluded that P-450 contributed only about 5% to ethanol metabolism; values were essentially unaffected by fructose. Thus, inhibition of cytochrome P-450 cannot explain the decrease in ethanol metabolism due to fructose in vivo. On the other hand, rates of methanol metabolism were decreased approximately 40% by fructose. The data are consistant with the hypothesis that by decreasing ATP, fructose prevents activation of fatty acids for generation of H_2O_2 via the peroxisomal Beta-oxidation pathway for catalase-dependent ethanol metabolism (Supported by AA-03624).

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I was pleasantly surprised and pleased to see an old friend [hairless-2 deermouse] on the cover of the No. 6 issue of **Peromyscus Newsletter**. However, I have some reservations concerning the recent finding of Hoppenhauer and Knapp who reported that the hairless-2 trait was incompletely dominant rather than recessive. Before finally establishing our colony of homozygous pairs, I maintained the hairless stock at Dugway for a number of generations using homozygous naked males and heterozygous females, and, of course, a supplementary colony to produce the heterozygous females. I don't recall a single instance among either segregating litters produced by the naked males X heterozygous females or litters of all heterozygous young, where genetics ot furred animals was distinguishable phenotypically. They all looked like the 10's of thousands of mice produced over the years in the normal colony. Is it possible that stock used in this test somehow became contaminated by a modifier?

One mutation we had that never got reported was what I believe was postjuvenal nude in the Pinyon Mouse, *P. truei*. Think I still have photos of it somewhere.

My project on the ecology of *Peromyscus* fleas in the Great Basin continues. Seems like it is going to last forever. Among several interesting discoveries was an undescribed species of flea on the Canyon Mouse, *P. crinitus*.

* * * * * *

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Our research centers on mammalian genetic and evolutionary problems which we are exploring using primarily laboratory bred and wild *Peromyscus*. The following are some of the current projects of interest:

I have long been interested in the origin of physiological and genetic mechanisms by which reproductive isolation arises, *i.e.* the actual basis of speciation in mammals. *Peromyscus* is an ideal group for these studies, because several instances of partial reproductive isolation are known. Most of our work has involved *P. maniculatus* and *P. polionotus* which will hybridize in captivity, although with extensive fetal and maternal mortality when *P. polionotus* is the mother. The hybrids exhibit pronounced reciprocal opposite heterosis for body size from fetal age to maturity. Placentas from reciprocal hybrids show more than a five-fold difference in mean weight. The basis for hybrid size and mortality effects is not clear although, to some extent seems to be attributable to progesterone influences on fetal-maternal histoincompatibility.
Most recently, we have examined the possibility that mitochondrial-nuclear genomic interaction in the species hybrids could be a source of dysgenesis, but combining mtDNA from one species with a nuclear composition 99% or more of the other, does not diminsih or exaggerate the hybrid phenomena (See PN# 3).

A recent hypothesis (Dover, 1983) concerning the possible basis for reproductive isolation between related species involves "molecular drive" or concerted gene evolution. Concerted changes in repeat DNA families over an evolutionary brief time may result in reproductive dysfunction of hybrids among population subunits, by analogy with hybrid dysgenesis in *Drosophila*. The effect may be mediated through transposition and/or gene conversion. The *L-1* (Long interspersed nuclear element #1) repeat family occurs widely in mammals, including *Peromyscus*. We are comparing a number of *Peromyscus* species and subspecies for concerted restriction site differences. Correspondence of restriction site changes with species, but not subspecific, boundaries would be consistent with the Dover hypothesis. We have two *P. maniculatus L-1* molecular probes homologous to the *Mus* MIF-1 fragment. We have made restriction maps of the probes, and sequenced most of one of them.

Another area of interest concerns comparative genetic linkage among rodents, concentrating on homology between *Peromyscus* and laboratory mouse, *Mus*. A number of interesting questions have escaped resolution, including whether linkages are conserved by natural selection; and the general question of the rate at which linkage decays over evolutionary time. In this connection we are mapping additional biochemical and coat color loci in the deer mouse.

A graduate student, Renee Flinchum, presented preliminary data from her dissertation study at the annual meeting of the Association of Southeastern Biologists. Her presentation was titled "A test of genetic drift theory in a small population of *Peromyscus polionotus*". Another student, Lisa Kwarsick, presented a paper on her work with Loren Knapp concerning high mortality in the hairless-2 mutant deer mouse.

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I am interested in six species of *Peromyscus* which occur on the 250,000 acre Sevilleta National Wildlife Refuge which is located about 60 miles south of Albuquerque. The six are *P. truei, difficilis, boylii, eremicus, leucopus,* and *maniculatus*. A seventh, *P. gratus*, is a possibility on the western part of the refuge. At the moment I am conducting sampling throughout the Refuge to gather basic data on habitat occurrence and abundance. Although at least five of the species occur in very close proximity, there does seem to be habitat segregation between most kinds, with at most two species existing syntopically. I plan to set up several large permanent study plots within which to mark animals so that spatial relationships of species and individuals can be examined. I'm interested in the degree to which morphology dictates habitat use and coexistence, and also in differences in life-history strategies and behavior which relate to uses of habitats of varying productivity, heterogeneity, and stability. I envision a long-term study. The Refuge is dedicated to research use, and has just been designated a Long Term Ecological Research site by NSF, with a group in the UNM Biology Department as the PI. We have large holdings of conventional museum specimens in the Museum of Southwestern Biology, and have, in recent years, augmented these with collections of karyotypes and frozen tissue suitable for electophoretic and other analyses. Three of us here, Jim Brown, Terry Yates, and I, are concerned with systematics, evolutionary biology, and ecology of small mammals, including *Peromyscus*.

* * * * * *

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I am investigating the proximate causation and functional significance of paternal behavior in the biparental California mouse, *Peromyscus californicus*. Laboratory studies focus upon the onset and maintenance of paternal behavior, the father's effects on growth, sexual maturation and reproduction of his offspring, hormonal and neural mechanisms underlying paternal behavior. We have just begun to study mate choice, kin recongition and monogamy in *P. californicus*.

We have found that the male's parental behavior is maintained postpartum by a chemosignal in his mates's urine. We are examining the chemical nature of the urine to determine what fraction and components of urine keep the male parental.

I intend to conduct comparative studies on other *Peromyscus* species that do or do not exhibit paternal behavior. I would appreciate hearing from anyone conducting field and/or laboratory studies on *P. eremicus* or *P. polionotus*.

* * * * * *

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Research Interest and Proposed Projects

Female-induced Pregnancy Block:

Over the past several years I have identified and studyed a reproductive block that occurs in female deermice (*Peromyscus*). Female *Peromyscus* fail to produce offspring when paired with a male from weaning to 150 days of age if an adult female or her odor is also present (97% effective). this phenomenon unlike the other forms of female-induced reproductive suppression known in mammals, is a postcopulatory process. Reproductively blocked females achieve puberty at a normal age, ovulate and copulate with the male. Embryo development is normal as is oviducal transport, however, the embryos do not implant.

My current research focuses on the physiological and endocrine bases of this phenomena. Specifically, I am evaluating the physiological and endocrine changes that occur in female white-footed mice (*Peromyscus leucopus*) during the preimplantation phase of pregnancy (Days 1-5). To date, these studies have shown that unlike other socially-mediated pregnancy blocks such as the male-induced "Bruce Effect", this pregnancy block is not the result of depressed prolactin levels. Likewise, stress is not involved as corticosterone level in blocked females are normal throughout preimplantation. Luteinizing hormone levels of blocked and unblocked females were also the same. Thus, pituatary signaling for corpus luteum formation is not affected. A number of other hormones are being assayed to expedite determining the endocrine mechanism(s) responsible for the female-induced preganacy block. Plans have also been made to test for embryo dysfunction via embryo transfer and to test for uterine receptivity via a deciduoma reaction.

The endocrine dimension of this line of research is dependent on the outcome of the experiments described above. However, the social and ecological implications of the pregnancy block will remain an open area of study for some time. The relationship between the pregnancy block and the social system of *Peromyscus* is one concern that I will pursue in the future. Initially, such studies would take place in enclosed populations. Eventually I believe that this phenomenon can be studied in natural populations.

Genetics of Reproductive Response to Photoperiod:

Individuals in some populaitons of *Peromyscus* are reproductively responsive to photoperiod while other individuals in the same population are not. This variability has been shown to have a significant genetic component and it has been shown to be amenable to artificial selection (Desjardins et al., 1986). Currently, Dr. Frank Bronson and I are selecting for strains of white-footed mice (*P. leucopus*) from a single population that are either reproductively responsive or unresponsive to photoperiod. At present, we are mating the F_2 generation animals of both strains to produce an F_3. The F_2 generations of both strains were 85-90% pure. We are optimistic that pure strains can be derived within a few generation of selection. The goals of this research are twofold. First, through appropriate crosses within and between strains we hope to be able to quantify the genetic basis of the variation in the reproductive response of these mice to photoperiod. Second, photoperiod is an important environmental cue in regulating seasonal reproduction, but not the only factor of importance. Upon the successful selection of these two strains of mice, we will be in a unique position to study the interaction of these other factors with photoperiod in strains of mice with a similar genetic background (both strains originating from a single population in Michigan). Thus, the potential exists in this research to begin to explain the multiple reproductive strategies found within natural populations.

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I am no longer doing research with *Peromyscus*, but keep the Newsletter active. It is a great contribution to this nobel genus.

My discoveries are now all old but include: 1) the winter aggregation in nests are family units; 2) this species frequently becomes torpid during cold snaps when the ground surface becomes frozen; 3) twice as many males as females make extensive innate (genetic controlled) dispersals even across unfavorable habitats when they become sexually active for the first time; 4) without ample nutritious food available they can succumb to "cold-weather-starvation," but will survive in live traps during freezing weather if provided with sufficient high-quality food; 5) the young of a litter may forage together with the adult male when the female is pregnant or has new young; 6) during winter the family groups may cache sizeable quantities of weed seeds, acorns, etc. in their nest site; 7) they can share a common tunnel with bumblebees but if only one nest chamber is available the first species to claim it gets it; 8) selection of mates is initiated anew each spring; 9) in the *P. maniculatus* population I studied at the UM George Reserve at least 4 to 10% of all matings were the result of brother-sister or parent-offspring matings; and 10) these rodents are probably the most delightful species of mammals known.

* * * * * *

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I have a colony of about 100 *Peromyscus polionotus*, mostly F_1 and F_2 offspring of wild-caught animals. About half of the animals are beach mice from Gulf and Bay Counties, Florida, and half are from 30 miles inland in Leon County, Florida. I am in the process of setting up a multigeneration experiment to study reaction norms of these populations in various environmental conditions.

* * * * * *

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Two major projects are currently in progress in this laboratory. First, the phylogenetic relationships of the *Peromyscus aztecus* assemblage of the *P. boylii* species group are being examined by protein gel electrophoresis. Data have been collected on electrophoretic homology at 32 structural loci among populations of *P. aztecus aztecus*, *P. a. evides*, *P. a. hylocetes*, *P. a. oaxacenmsis*, *P. spicilegus*, and *P. winkelmanni*. Homology data will be obtained on over 40 loci when this project is completed. The electrophoretic homology within the *P. aztecus* assemblage is being standardized against the electrophoretic variation within the *P. boylii* assemblage reported by Rennert and Kilpatrick (1986, 1987). this standardization will allow us to examine the phylogenetic relationships of the entire *P. boylii* species group.

Second, a DNA-DNA hybridization laboratory is being equipped and a "DNAlyzer" is being constructed. This laboratory should be operational by the middle of April. DNA has been isolated and purified from 24 taxa of peromyscine-neotomine rodents. The thermal stability of single-copy DNA-DNA hybrids among pairs of these 24 taxa will be examined to allow inference of the phylogenetic relationships among these peromyscine rodents.

I am building a collection of single copy DNA from rodents and would appreciate additional samples. DNA can be isolated from liver, spleen, kidney, heart, brain, muscle, etc that has been frozen or preserved in ethanol at room temperature. Please write or call if you could provide this collection with tissue from any rodent species and I will provide you with directions and materials for preserving these tissues.

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Behavioural Changes in Wild, *Trichinella*-Infected Rodents.

Trichinellosis is one of the most widespread and clinically important parasitic zoonosis on the European and North American continents. The most common aetiological agent of this disease, *Trichinella spiralis*, is acquired through the ingestion of the undercooked flesh of a wide variety of domestic and wild mammals which can harbour the larvae of this parasite. The recent discovery in North America of *T. pseudospiralis* (Wheeldon et al., 1983, J. Parasit. 69(4):781-782) which can develop both in mammals and birds,underscores the plea of Zimmermann (1977, In Parasitic Diseases of Wild Mammals, J.W. Davis and R. G. Anderson (eds.). Iowa State Univ. Press, Ames, Iowa, pp. 127-139) for intensive studies of trichinellosis in wildlife. Whereas conventional approches to such studies (Rausch et al., 1956, J. Parasit. 42:259-271; Schad et al., 1984, J. Parasit. 70:372-377) have provided insight into the distribution of the parasites within various host species, they have yielded little information concerning the transmission dynamics of these infections in the field. Indeed, such studies have raised a perplexing problem regarding the source of infection of many small predators which feed primarily on rodents (Zimmermann,

1971, op. cit.). Thus, while the prevalence of infection is high among the predators, their prey is very rarely found to be infected. The work of Rau (1983, Parasit. 86:311-318, 319-322; 1984, Parasit. 88:415-419;1985, J. Parasit. 71(6):774-778) and Zohar and Rau (1984, J. Parasit. 70(6):927-930) on the behavioural changes and competitive fitness of laboratory mice infected with *T. spiralis* and *T. pseudospiralis* suggests that these infections predispose such rodents to predation. Conceivably, wild rodents bearing *Trichinella* infections may be similarly affected and may be removed rapidly from the population, thus transmitting the infection to predators. Consequently, infected rodents would rarely be encountered in the field. Their apparent absence from the rodent population may thus be an artifact of their fast passage through the system.

The study under way is addressing the behavioural changes induced by the parasites in wild rodent hosts and their effects on the susceptibility of rodent hosts to predators. More specifically, the study measures the impact of trichinellosis on the circadian activity rhythms of *Microtus pennsylvanicua* and *Peromyscus maniculatus* in infrared beam activity chambers. In order to assess susceptibility topredation, infected and control animals are exposed to American kestrels (*Falco sparverius*) and screech owls (*Otus asio*). Furthermore, to determine whether carrion feeding may expose wild rodents to infection with *Trichinella*, visits of small mammals to baiting stations are monitored by means of multicapture enclosures. The study will provide some insight into the events accompanying the transmission of trichinellosis under field conditions, with specific reference to the role of selected small, wild mammals.

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At ODU, I have a proposal pending with the Virginia Department of Game and Inland Fisheries to study the status and distribution of the Pungo mouse, *Peromyscus leucopus easti*. This mouse is known only from the brushy thickets of three locations in extreme southeastern Virginia. Its possible restriction to this habitat type and its possible distribution in similar coastal havitats to the north and south are unknown. Mike Carleton of the Smithsonian has agreed to examine specimens. The non-game program in Virginia may be interested in supporting a study to determine the status of the cotton mouse, *Peromyscus gossypinus*, in the Dismal Swamp region. This mouse has not been seen since the studies of Lee Dice and Don Hayne in the 1930's (which led to their often-cited paper on interbreeding between cotton and white-footed mice).

John Rose, who is trying to raise money for college expenses, is willing to trap and ship local meadow voles, cotton rats, rice rats, or white-footed mice to researchers who might need specimens from eastern Virginia.

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We have begun a genic investigation of the systematic relationships among the three subspecies of *Peromyscus maniculatus* that occur in the eastern United States. *P. m. bairdii* is a short-tailed mouse of low elevation hayfields and open grasslands from Pennsylvania and New York south and westward. *P. m. nubiterrae* and *P. m. gracilis* are long-tailed mice found in high elevation forests. The current range of *P. m. gracilis* is restricted to formerly glaciated regions of northeastern Pennsylvania and surrounding states, whereas the range of *P. m. nubiterrae* corresponds to unglaciated regions from central Pennsylvania to northern Georgia. Although our study is in a preliminary stage, data suggest that there may be a separation, based on overall genic similarity, between the long-tailed subspecies and *P. m. bairdii*. Short-tailed *P. m. bairdii* from Kansas also appear to be mor egenically similar to *P. m. luteus* from Texas than to *P. m. bairdii* from Pennsylvania. We are continuing to collect representative samples of the subspecies of *P. maniculatus* from various localities in the eastern United States, and these will be added to our initial data set.

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I am now into my ninth consecutive year of studying the population and behavioral ecology of *Peromyscus leucopus* and *P. maniculatus* at the Mountain Lake Biological Station in southwestern Virginia. Our main focus now is to conduct field and laboratory studies to determine the adaptive significance of infanticide in these species and to determine its demographic consequences with respect to population dynamics. Donna Cicirello (currently at Michigan State University) and I have a paper in press in Animal Behaviour that shows that adult males that have sired pups within the previous 30 days will not kill any pups within their own home ranges, whereas dispersing males and/or males that have not mated within the previous 30 days do kill strange infants. Females usually kill any strange pups they encounter that are not in their own nests. Field and laboratory evidence suggests that prior social experience is important in determining if, where, and when an individual will commit infanticide. We are currently attempting to determine the effects of density on the occurrence of infanticide by males and females.

Peromyscus bred throughout the 1988-89 winter at Mountain Lake. This is the second time in the last eight years that mice have bred during winter. The previous time was in 1985-86. Winter breeding in both situations coincided with a large autumn mast crop. This anecdotal evidence suggests that large spring breeding populations and subsequent high densities may result from an abundant overwinter food source which in turn makes winter breeding possible. The role of acorns in the breeding biology of small seed-eating rodents and even larger species such as deer, bears, and turkey warrant further investigation.

Recent Publications:

Krohne, D.T., J.F. Merritt, S.H. Vessey, and J.O. Wolff. 1988. Comparative demography of forest Peromyscus. Can. J. Zool., 66:2170-2176.

Fitsgerald, V.J. and J.O Wolff. 1988. Behavioral responses of escaping Peromyscus leucopus to wet and dry substrata. J. Mammal., 69:825-828.

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Brain size in captive versus wild populations of *Peromyscus maniculatus bairdii*

Relative brain size (brain mass/body mass) in rodent species is suprisingly labile both within and among generations. Two approaches have been taken to understand this variation; one ecological/evolutionary (E/E) and one developmental/psychological (D/P). Mace and Eisenberg (1982) represent the former approach, claiming that brain size in any particular deer mouse species changes, in part, in response to the number of other deer mouse species that occur in the same geographic range or habitat. In sympatry it is presumed that evolutionary selection pressures have led to large brains since each species must store and process the information necessary to compete with the others for food and nest sites. When a species from an area of sympatry is compared to the same species in an allopatric area, the average brain size is usually larger in sympatry. It follows therefore that if a species is removed from all contact with other species with which it most likely competes, for sufficient time, selection on brain size may relax and brain size may decrease.

The second factor that influences brain size is the amount of environmental complexity that a developing animal experiences (Renner and Rosenzweig 1987). Structurally and/or socially impoverished or enriched living conditions can decrease or increase relative brain size by as much as 10 percent. It follows that if an animal is removed from the wild and allowed to rear its young in an impoverished lab cage the young should become relatively small-brained adults. Contrary to the E/E hypothesis, a brain size decrease can be realized within the lifetime of an individual. I have been measuring the relative brain sizes of a closed laboratory colony of prairie deer mice (Peromyscus Stock Center-BW stock) and comparing them to individuals of the same species that were wild-caught in Michigan at about the same time the lab colony was established (early 1940's). The brains of mice recently collected from the colony are significantly smaller than the brains of individuals collected in the 40's at the site where the lab colony founders were originally collected ($F=13.23$, $P=.0005$). However, the observed reduction in brain size may have resulted from either 1) the intergenerational relaxation of selection expected if the E/E hypothesis is correct or, 2) may merely be a within-generational effect produced by the simple, impoverished environments that each generation of young mice experience growing up in the lab.

Presently I am seeking funds (or collaborators in Michigan) to collect additional data to distinguish between these competing hypotheses. To do so I must have two data sets; the brain sizes of wild mice living today in the area where the laboratory founders were collected around 1940, and brain sizes of mice that were raised in the lab to wild-caught mothers. The lab colony that I have been working with was derived from individuals collected in Washtenaw Co. MI and was kept at the University of Michigan's Museum of Vertebrate Zoology (MVZ) until it moved to its present location at the PSC. Unfortunately, neither the curatorial staff at the MVZ nor the PSC are aware of any lab mice that were preserved during the course of the colony's long history. Nor does the Museum have skulls from recently collected wild mice with which I could compare the lab and 1940's- collected wild individuals. Therefore, I plan to collect Washtenaw County mice and breed them in captivity. Their first litters will be raised until adulthood at which time parents and offspring will be sacrificed and cranial volumes will be measured. My expectation is that the brain sizes of recently collected mice will not differ from those of field-caught animals from the 1940's. If this is true and if after only one generation in the lab brain sizes have decreased to the size of individuals from the long-term captive population, I can exclude

the E/E hypothesis and presume that the effect is due solely to lack of environmental complexity in the laboratory. If, however, the brains are no different than those of their wild-caught parents the E/E hypothesis remains inteact. Finally, if brains of lab-raised mice from wild-caught mothers are smaller than wild-caught mouse brains but not as small as those of long-term lab-raised individuals then it is possible that either both factors are acting on brain size or (less likely) that impoverishment can have cumulative effects over multiple gererations.

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