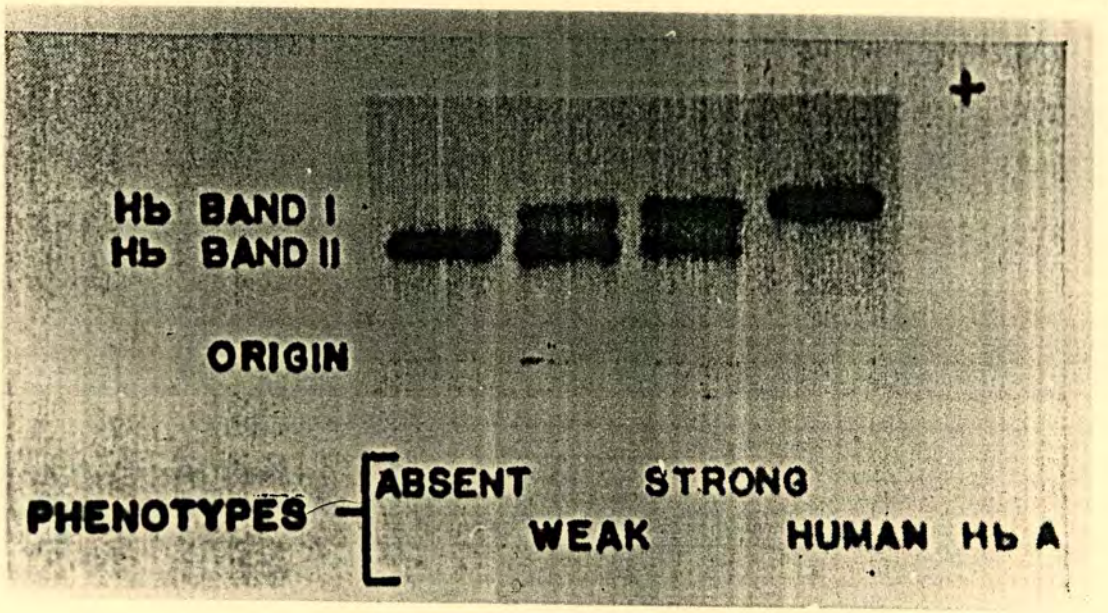
PEROMYSCUS NEWSLETTER

NUMBER EIGHTEEN



SEPTEMBER 1994

Cover: Starch gel electrophoresis phenotypes of deer mouse hemoglobin stained with amido black, one of the earliest published photographs of *Peromyscus* electrophoretic patterns. (From: Rasmussen, DI, JN Jensen & RK Koehn. 1968. *Biochem. Genetics* 2:p.89. Permission Plenum Press)

PEROMYSCUS NEWSLETTER NUMBER 18 ---

Our regrets for being a few months behind schedule with this ("September") issue. We expect to be back on a regular schedule by the March Issue.

In this issue we feature the first of two historical accounts of **"Peromyscus and Electrophoresis"**, and the role this technology played in advancing *Peromyscus* research. The two-part narrative will be divided into "the early years" (1960-1970) and "the heyday" (1971 - 1985). Combined with this first account we recognize the achievements of two "Peromyscus Pioneers", Charles Foreman and David Rasmussen, who were among the first to employ electrophoresis with *Peromyscus*. The second installment of the electrophoresis story will appear in *PN* #20. We trust our readers will enjoy these reviews. In the future, we plan to have a similar series on "Cytogenetics and *Peromyscus*."

Also in this issue, we have the updated list of established genetic loci in various *Peromyscus* species. Descriptions of several new genes have been published since our previous listing in *PN* #14 (September 1992).

As always, we appreciate the accounts of on-going research submitted for our "Contributions" section. Remember we also will publish announcements relevant to *Peromyscus* and related genera. We consider the scope of the Newsletter to include not only Genus *Peromyscus*, but other peromyscine rodents as well.

The *Peromyscus* Genetic Stock Center encourages use of deer mouse coat color mutations wherever feasible. Nearly half of the budget of the Stock Center is devoted to maintaining mutant stocks, particularly the 20-plus coat color variants. As cost of maintaining mutant stocks continues to increase, justification for maintaining them becomes critical. Readers who would like to obtain any of the color variants should please contact the Stock Center. These animals are useful for classroom demonstrations of Mendelian genetics or exhibits. Lifespan often exceeds three years, so they can be kept through several semesters. Flat skins of the color mutants are also available for a modest fee.

PN and the *Peromyscus* Stock Center has a new E-mail address:

peromyscus@stkctr.biol.scarolina.edu

Deadline for entries for the next issue is 15 March 1995.

PEROMYSCUS NEWSLETTER is produced by the

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NEWS, COMMENT and ANNOUNCEMENTS

Bruce Buttler has a new bibliography available - a listing of references on *Peromyscus* as prey species. This is part of a continuing series of *Peromyscus* bibliographies compiled by Bruce. Previous ones cite the literature on reproduction, genetics and ecological monitoring. These may be obtained by writing Bruce at Canadian Union College, Box 430, College Heights, Alberta T0C 0Z0, CANADA.

We received a warm letter from **T.C. HSU.** Dr. Hsu has retired, but tells us he is still working part-time on human cancer problems. He also says he has pleasant memories of his "days trapping and playing with these [Peromyscus] wonderful creatures." T.C. Hsu was one of our "Peromyscus Pioneers" (See Issue # 8).

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REQUEST

Search for Investigators Performing Occupational Studies with Hantavirus

We are in search of investigators who would be interested in testing serum samples for previous Hantavirus exposure. Approximately ten researchers from the University of Arizona who actively perform field research with rodents have had serum samples taken for the purpose of obtaining baseline values. At the present time the Arizona States Health Department will only test individuals for Hantavirus who are actively sick or deceased. Alternatively, if any researchers who are currently studying the Sin Nombre Hantavirus and can provide us with either antigen or clones of the virus so that we may establish our own occupational test would also be welcome. **Any information or suggestions of contact persons that *PEROMYSCUS NEWSLETTER* readers can provide is greatly appreciated.** Please contact:

Sarah Palmer (Research Coordinator) or David Mount (Chairman)
Institutional Biosafety Committee
Life Sciences South
University of Arizona
Tucson, AZ 85721
(602) 621-3441

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We received a note and newspaper clipping from **W. Newman Bradshaw** *regarding Hantavirus, now residing in Sierra Vista, AZ. He remarks that Hantavirus is still of much local concern. Dr. Bradshaw was formerly on the faculty at the West Virginia University and conducted some of the early cytogenetic studies with* Peromyscus.

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A communication from **Jorge Fernadez Hernandez,** Director of the National Center for the Breeding of Laboratory Animals (CENPALAB), Havana, Cuba, reminds us that the facility focuses on the principal lab animal species, experimental toxicology and biotechnology.

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A note from **Cheri Jones,** Denver Museum of Natural History: She tells of several recent publications treating the biology of *Podomys (=Peromyscus) floridanus*. (See "Recent Publications" p. 39 this issue *PN*)

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Twenty-eight papers and posters dealing primarily with *Peromyscus* were presented at the 1994 Annual Meeting of the American Society of Mammalogists. The meeting was hosted by the Smithsonian Institution in Washington, DC, June 18-23.

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A color photo of a deer mouse was featured on page one of the July 11th issue of The Scientist. The publication carried a feature article on how the CDC and other scientists around the U.S. cooperated to quickly identify the hantavirus responsible for the "Four Corners disease" outbreak last year, and how Peromyscus maniculatus was found to be the primary viral host.

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Virginia Hayssen has relocated to the Department of Physiology and Environmental Science, University of Nottingham, Sutton Bonongton Campus, Leicestershire, UK. She has established a *Peromyscus* colony there, which is one of the very few outside North America.

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Inbreeding and inbreeding avoidance in *Peromyscus* continues to be of interest in both laboratory and field studies. A 14 October 94 paper in *Science* by **Julie Jimenez, Kim Hughes, Glen Alaks, Laurie Graham** and **Robert Lacy** of Chicago's Brookfield Zoo demonstrates detrimental effects of only a few generations of inbreeding in experimental populations. **David Ribble, Jerry Wolff** and others have published on the topic during the past year or two. It is becoming clear that the *Peromyscus* genome is sensitive to destabilization by inbreeding, and dispersal behavior by young reinforces outbreeding in natural populations.

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A recent article in J. Mammalogy *(1994. 75:704ff) Paul Stapp and co-workers at Colorado State University found that fluorescent powders, now popular for tracking animals, produced no serious pathological effects in* P. maniculatus, *provided that exposure of respiratory tissues of study animals was minimal.*

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POSTDOCTORAL POSITION IN EVOLUTIONARY PHYSIOLOGY

Postdoctoral research associate needed for study of natural selection on whole-animal physiological performance of deer mice. Position starts 1 March 1995 or as soon thereafter as possible, but must start by 1 May 1995. Applicants should have a Ph.D. in physiological ecology or a related field. Desirable qualifications include experience with handling and breeding small mammals, computerized data acquisition, whole-animal physiological studies, and radio-tracking, and an interest in evolutionary physiology. Must be in excellent physical condition to work (about 6 months/yr) at a remote high altitude (3,800m = 12,500 ft) research station near the California-Nevada border. Spouses and dependents are not allowed to reside at the field station. A commitment to follow appropriate safety procedures for handling mice that may harbor Muerto Canyon virus (hantavirus) is required. The University of Nevada-Reno is an AA/EO employer.

To apply:

By 15 January 1995, send a cv and the names, phone numbers, and addresses (including email addresses, if available) of three references to:

Dr. **Jack Hayes**, Department of Biology, University of Nevada, Reno, NV 89557 (jhayes@scs.unr.edu)

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POSTDOCTORAL POSITION TO STUDY ECOLOGICAL AND ENVIRONMENTAL CORRELATES OF HANTAVIRUS DISTRIBUTION AND ABUNDANCE

Postdoctoral research associate needed to work on ecological aspects of NIH-funded study Muerto Canyon virus (hantavirus). Position starts Jan 1, 1995 or as soon thereafter as possible. Field experience with small mammals (preferably in the western US), strong quantitative (statistical) skills and a Ph.D. are required. Experience in small mammal ecology and physiology, virology, epidemiology, ectoparasites, or GIS are desirable. Applicants must be willing to travel extensively and to interact with a diverse group of scientists. Responsibilities include assisting with design and implementation of field studies to assess the distribution and abundance of Muerto Canyon virus in small mammal communities, primarily in western Nevada. Successful applicant will use varied analytical tools to search for ecological and environmental factors that may explain the distribution and abundance of the virus. He or she will be required to follow appropriate precautions for dealing with wild mammals that may harbor hantavirus, and must submit and NIH postdoctoral fellowship application within 1 year of start of position.

To apply, send a cv and the names, phone numbers, and email addresses of three references to Dr. **Jack Hayes**, Department of Biology, University of Nevada, Reno, NV 89557 by 15 December 19994. For additional information contact Jack Hayes at jhayes@scs.unr.edu or 702-784-6076.

The University of Nevada is an Equal Opportunity/Affirmative Action Employer

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HANTAVIRUS UPDATE

The outbreak of a previously unrecognized type of *Peromyscus*-transmitted hantavirus during 1993, appears to have moderated considerably this year. Only 29 verified new cases in the U.S. with 12 deaths were reported during the first 11 months of 1994 compared with 53 and 32 deaths during the previous year. The incidence of this year's cases has no clear focus analogous to the concentration of cases in the Four Corners region in 1993. Nevertheless, there is concern that more cases have been reported from the eastern states, and that North American rodent species other than the deer mouse may also transmit hantavirus infections. *P. leucopus* positive for hantavirus have been found by Mike Tannenbaum and others. J-W Song and colleagues (*Lancet* 344:1637. 1994) have associated a hantavirus genetic type from *P. leucopus* with a human case from New York.

Some items of interest:

Eastern U.S. human hantaviral cases were reported from New York, Indiana, Virginia, Florida and, perhaps, Pennsylvania. There are also three cases reported from western Canada.

934 biologists and others who were at risk of exposure through handling of deer mice and other small rodents were sampled at the annual meeting of the American Society of Mammalogists and at other sites. Eight mammalogists tested positive for antibody to hantavirus. One of these had suffered hantaviral pulmonary syndrome previously. All others were negative.

Terry Yates of University of New Mexico has a CDC contract to sample *Peromyscus* populations nationally for hantavirus. The results of this study should soon be available.

Frozen tissues of *Peromyscus*, some collected more than a decade ago, have tested positive for the virus, and tissue samples from patients exhibiting symptoms have also tested positive. At least eight cases have been verified which occurred before 1993. This is further evidence that the "new" hantavirus in *Peromyscus* may not be recently evolved.

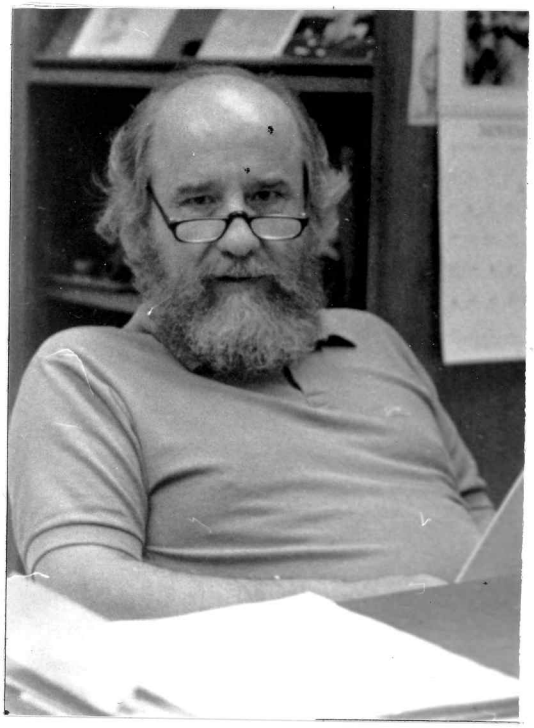
The causative agent of hantaviral pulmonary syndrome (HPS) has undergone yet another name change from FCV (Four Corners Virus) to MCV (Muerto Canyon Virus) and now, most recently, to SNV (Sin Nombre Virus). Maybe SNV should be "several names virus"!

The hantaviral pulmonary disease associated with SNV is about 10 times more deadly than syndromes caused by other hantaviruses.

We have an unconfirmed report of two wild-caught *P. leucopus* which were caged together for more than a year, then tested for hantavirus antibody. One tested positive while the other tested negative, suggesting that adult mouse-to-mouse transmission may not result from simple association.

MMWR 43/No.RR-7 (May 13,1994) contains a supplement "Laboratory Mamangement of Agents Associated with Hantavirus Pulmonary Syndrome: Interim Guidelines". Included is a table of biosafety standards for handling host species.

Charles Foreman



David Rasmussen

PEROMYSCUS PIONEERS:

CHARLES W. FOREMAN and **DAVID I. RASMUSSEN**

PEROMYSCUS AND ELECTROPHORESIS:

THE EARLY YEARS 1960 - 1970

The application of electrophoresis to the study of evolution and systematics brought about a revolution in *Peromyscus* research and greatly increased knowledge of population genetics in the genus. In fact, the dynamic activity of the 1930s and '40s resulting from the work of Lee Dice, Frank Blair and their contemporaries had largely subsided by the late 1950s. A general impression existed that little new significant information could be obtained utilizing the traditional morphological approaches then available. The number of investigators actively working with *Peromyscus* genetics had diminished to fewer than a dozen. Then, suddenly, by the mid-1970's several major groups, representing numerous biologists and their students, were enthusiastically sampling many species of the genus for protein polymorphisms. By 1978 reports of "biochemical" (= protein electrophoretic) variation within *Peromyscus* were frequent in such publications as *Evolution* and *Journal of Mammalogy*. This new technique made it feasible to address questions in evolutionary genetics and systematics which could not be resolved by the previously existing methodology.

How did this remarkable transformation in *Peromyscus* evolutionary research come about? And who were the pioneers who brought it about? Here we recognize the parallel careers of two individuals who were among the first to use electrophoresis in *Peromyscus*: Charlie Foreman and David Rasmussen. They, along with Murray Johnson, Charles R. Shaw and a few others, led the first wave of electrophoresis research with the genus. Much of this early work with natural populations involved hemoglobin and serum proteins, especially albumin and transferrin. These initial forays were followed during the 1970s and early '80s by a stampede of activity in which many *Peromyscus* populations and individuals were sampled for numerous tissue enzymes in addition to blood proteins. We will defer a discussion of this heyday of "*Peromyscus* and Electrophoresis", for a subsequent issue of *PN*.

Charles Foreman was born in Norman Park, Georgia, November 2, 1923. During World War II he served in the U.S. Army as an aircraft electrician, a pre-engineering student at the University of Georgia and a pre-medical student at Vanderbilt University. After the war Foreman completed his undergraduate education at the University of North Carolina in 1949, and received his MA (1951) and Ph.D. (1954) in Physiology from Duke University Medical School. His disseration research, subsequently published in *J. Cell. Comp. Physiol.* (1954. 44:421*ff*), described oxygen dissociation profiles of mammalian hemoglobin in various wild species, including *Peromyscus*. After brief faculty appointments at Wofford College in South Carolina and the University of Maryland, in 1956 he joined the faculty of Pfeiffer College, a small private school in western North Carolina where he remained until 1963 when he moved to the University of the South at Sewanee, Tennessee. He spent the rest of his academic career at Sewanee.

In 1960 Charlie visited the Biology Division of the Oak Ridge National Laboratory (ORNL) as a summer research associate to continue his hemoglobin studies with Dr. Raymond A. Popp. This coincided with Popp's first efforts to identify hemoglobin differences between laboratory mouse (*Mus*) strains using electrophoresis. Foreman subsequently was granted a contract permitting him to use the ORNL facilities as needed. Electrophoresis was just then coming into its own as a laboratory separatory method. Foreman recognized that electrophoretic migration of hemoglobin might differ among mammalian species and had potential taxonomic value. In 1960 Foreman published the first paper to describe protein electrophoretic variation within a *Peromyscus* species, as well as differences among *Peromyscus* species and other mammals (*Am. Mid. Nat.* 64:177*ff*). He noted that two major hemoglobin bands, and sometimes three, were apparent on paper strips, which differed from the typical single band and diffuse types observed in laboratory mice. Perhaps more significantly, Foreman found slightly different patterns of electrophoretic mobility when comparing *P. leucopus* or *P. gossypinus* with *P. maniculatus* or *P. polionotus*. About the same time Murray Johnson and Merrill Wicks (1959. *Syst. Zool.* 8:88*ff*) recorded differences in electorphoretic patterns of serum proteins among mammalian species, including those of the genus *Peromyscus*, but they did not note within-species polymorphism.

Meanwhile, at the University of Michigan, David Rasmussen was completing his doctoral work under the direction of Morris Foster, a mammalian geneticist. David's dissertation research was a population genetic study of *P. maniculatus* using a two-allele erythrocytic antigen system (1961. *Genet. Res. Camb.* 2:449*ff*). The study, subsequently published in *Evolution* (1964. 18:219*ff*), estimated inbreeding coefficients for a Michigan deer mouse population. This study won Rasmussen the prestigous A. Brazier Howell Award from the American Society of Mammalogists. In 1962, when David finished his degree, the Michigan mammalian genetics group, including Shaw, Foster and Elizabeth Barto, was still actively working with *Peromyscus*. The 1960 Foreman paper was of considerable interest to this group, and was surprising to some extent because it originated with an individual who was not well known among mammalian evolutionary geneticists and who was located at a small, obscure college in southern Appalachia. Nevertheless, the potential utility of electrophoresis to detect protein polymorphisms in natural populations was immediately evident.

David Rasmussen was born 11 December 1934 in Ogden, UT. His father, D. Irvin Rasmussen, was a well known wildlife biologist at Utah State University at Logan and the U.S. Forest Service, thus David grew up with a feeling for mammalogy. He obtained both the B.S. (1956) and M.S. (1958) degrees at the University of Utah and then moved on to Ann Arbor. After completing his doctorate in 1962, he spent a year at UC Berkeley as an NIH postdoctoral fellow before joining the faculty of Arizona State University, where he remained until retirement. Upon arrival at at ASU, David, with his graduate students Richard Koehn and Neil Jensen, initiated a program to explore the genetics and distribution of electrophoretic variants of serum transferrin, (1966. *Genetics* 54:1353*ff*), albumin (1971. *J. Mamm.* 52:508*ff*), and serum esterases (1971. *Comp. Biochem. Physiol.* 39B:19*ff*). Rasmussen also authored the "Genetics" chaper in J.A. King's *Biology of Peromyscus* (1968) and provided the line drawings for chapter heads in the volume.

In the early 1960's, at Michigan, Charles Shaw, a medical doctor by training, with specialties in oncology and child psychiatry, had become interested in the genetics of electrophoretically-identified isozyme variants of lactate dehydrogenase (LDH). Clement Markert (1963. *Science* 140:1329*ff*), at the time, had recently proposed that electrophoretic patterns of LDH variants observed during mouse development could be explained as tetramers of two gene products. In the deer mouse stocks at Michigan a genetic variant for the B-subunit of LDH was discovered by Shaw and Barto (1963. *PNAS* 50:211*ff*) providing support for the Markert model (See "Peromyscus Pioneer: Elizabeth Barto" in *PN*#7). Subsequently, Shaw and Barto (1965. *Science* 148:199*ff*) also described inheritance of autosomally inherited glucose-6-phosphate dehydrogenase in *P. maniculatus*. Graduate students at Michigan during the 1960s also adopted electrophoresis to study protein polymorphism in *Peromyscus*. Sherman Randerson, analyzed the genetics of erythrocytic esterase variants in the deer mouse (1965. *Genetics* 52:990*ff*), while James Brown and Carl Welser surveyed 14 species of *Peromyscus* for serum albumin variation and formally analyzed the genetics of albumin using *P. maniculatus* X *P. polionotus* species crosses (1968. *J. Mamm.* 49:420*ff*).

During this same period, Foreman, by now at Sewanee, mapped hemoglobin tryptic peptide patterns in six *Peromyscus* species using two-dimensional electrophoresis/ chromatography (1964. *J. Cell Comp. Physiol.* 63:1*ff*), and with acetate strip electrophoresis analyzed the formal genetics of hemoglobin variation in *P. gossypinus* and *P. leucopus* X *P. gossypinus* crosses (1966. *Genetics* 54:1007*ff*). He also surveyed 10 species for hemoglobin electrophoretic type (1968. *Comp. Biochem. Physiol.* 25:727*ff*). Independently, Rasmussen, Koehn and Jensen were using starch gel electrophoresis to examine hemoglobin inheritance in *P. maniculatus* (1968. *Biochem. Genet.* 2:87*ff*), and Robert Thompson and several associates at the University of Mississippi reported a survey of *P. maniculatus* subspecific electrophoretic variation in hemoglobin hemolysates (1966. *Nature* 210:1063*ff*). The latter workers were also the first to separate fetal hemoglobin from deer mouse. Rasmussen and Foreman had proposed somewhat conflicting models to explain the single, double and triple band electrophoretic hemoglobin phenotypes of *Peromyscus*. Since whole hemoglobin molecules were involved, rather than globin subunits, at that time it was not clear whether one or both globin chains were involved in the observed variation. They attempted to reconcile their views with limited success during a personal conference at the 1968 meeting of the American Society of Mammalogists at Nags Head, NC. The issue was eventually resolved more than a decade later by Lee Snyder at Stanford (1978. *Genetics* 89:511*ff*) and Richard Padgett, Marshall Edgell and others at the University of North Carolina (1987. *Mol. Biol. Evol.* 4:30*ff*).

It was also at the Nags Head meeting and later in a *Bioscience* article (1969. 19:418*ff*) that Rasmussen questioned some of the studies which utilized electrophoresis in systematics. He correctly pointed out that electrophoretic (or immunological) variability of proteins, in the absence of knowledge of the underlying genetics or the within-taxon variation, was inappropriate for phylogenetic inference, and constituted a typological approach to taxonomy. While Rasmussen did not specifically cite offenders, the studies published by Murray Johnson (1968. *Syst. Zool.* 17:23*ff*) and Michael Petersen (1968. *Am. Mid. Nat.* 79:130*ff*) were cases in point which involved *Peromyscus*. Later, Bowers *et al.* (1973. *Evolution* 27:378*ff*) disputed Rasmussen's conclusions (1969. *Symp. Zool. Soc. Lond.* 26:335*ff*) regarding *P. maniculatus* protein polymorphism in Arizona, claiming that some unrecognized *P. melanotis* populations were included in the study. At the time of the study the latter species was known only from

Mexico. Thus, Rasmussen was at the center of some of the early skirmishes which arose from electrophoresis research. The opening shots of the neutralist-selectionist debate were being fired by J.L. King and Thomas Jukes (1969. "Non- Darwinian Evolution", *Science*, 164:788*ff*) and Motoo Kimura (1968. *Nature* 217:624*ff*) at this time. This controversy, which emanated from Richard Lewontin and J. Hubby's (1966. *Genetics* 54:595*ff*) study reporting high levels of allozyme heterozygosity in *Drosophila* populations, reverberated through *Peromyscus* population genetics during the next decade. Charlie Foreman, although not a dogmatic advocate of the neutralist view, was among the first to call attention to it with reference to *Peromyscus* in presentations at the Southeastern Biologists' annual meetings and elsewhere during the late 1960's.

By 1970 investigators elsewhere were beginning to use electrophoresis for analysis of *Peromyscus* genetics, *e.g.* David Cameron and Alan Birdsall (1970. *Biochem. Genet.* 4:655*ff*; *Genet. Res., Camb.* 16:355*ff*), Charles Biggers (1971. *J. Mamm.* 52:376*ff*) and Kenneth Griswold (1971. *J. Hered.* 62:339*ff*). At the same time a collaboration between Michael Smith of the Savannah River Ecology Laboratory and Robert Selander, then at the University of Texas, was underway to survey electrophoretic variation in numerous enzymes in populations of *P. polionotus* (1971. *Univ. Tex. Publ.* 7103:49*ff*). The latter was the first of many such extensive studies of polymorphism and heterozygosity in *Peromyscus* during the 1970s, and it heralded the "golden age" of allozyme studies of species within the genus.

Charlie Foreman phased out his *Peromyscus* stocks about 1972 to devote more attention to his teaching responsibilites. He eventually became chairman of the Biology Department at Sewanee, and served several years in that capacity. David Rasmussen's research focus turned from mammals to fish and insects during the '70s. His final paper on *Peromyscus* (1976. *Biochem. Genet.* 14:541*ff*) was a joint effort with his former student, Neil Jensen, and a graduate student, John Merkle, describing multiple hemoglobin loci in deer mice and chipmunks. David was the first to formally define an immunological locus (*Pm* blood group) in *Peromyscus* and also described the transferrin (*Trf*) locus in deer mouse. In 1966 Foreman first described by formal genetics a hemoglobin locus (*Hb*) in *Peromyscus* in *P. leucopus*, and Rasmussen described the same locus in *P. maniculatus* soon afterward. Thus, they were true pioneers in *Peromyscus* biochemical genetics research.

Charlie and David both retired last year. Charlie resides in Sewanee with his wife, Betty. One daughter, Rachel, is a cat veterinarian in Bellingham WA, the other, Millicent, a teacher in Winston-Salem NC. and their son, Roy, is a sergeant in the U.S. Army. David and his wife, Deon, live in Tempe. Their four children pursue diverse lives. One daughter, Jan, lives at home; another daughter, Lora, is a secondary school science teacher in Verde Valley, AZ; one son, Tab, is a Professor of Anthropology at Washington University, St. Louis; and another son, Brian, is a graduate student in Landscape Architecture at the University of Arizona, Tucson. During the summer months, Lora and Brian are guides on western rivers. Tab Rasmussen was recently in the news as a consequence of his discovery of fossils of a new species of early hominid.

We wish Charlie and David well in their retirement. We continue to recognize and appreciate them for their early achievements and for forging the path into a new era of *Peromyscus* research for biologists who followed.

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 Special Projects 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 **$10 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 fee. Arrangements for special orders will be negotiated. 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. polionotus leucocephalus (LS Stock)	Derived from beachmice wild-caught on Santa Rosa I., FL. and bred by R. Lacy. Fourth to seventh generation in captivity.
P. leucopus (LL Stock)	Derived from 38 wild ancestors captured between 1982 and 85 near Linville NC. Eighth to tenth generations in captivity.
P. californicus insignis (IS Stock)	Derived from about 60 ancestors collected between 1979 and 87 in Santa Monica Mts. CA. Seventh to tenth generation in captivity.
P. aztecus	Derived from animals collected on Sierra Chincua, Michoacan, Mexico in 1986 Fourth to seventh generation in captivity.
P. maniculatus X *P. polionotus* F_1 Hybrids	Sometimes available.

MUTATIONS AVAILABLE FROM THE STOCK CENTER[1]

Coat Colors	ORIGINAL SOURCE
Albino *c/c*	Sumner's albino deer mice (Sumner, 1922)
Ashy *ahy/ahy*	Wild-caught in Oregon ~ 1960 (Teed *et al.*, 1990)
Black (Non-agouti) *a/a*	Horner's black mutant (Horner *et al.*, 1980)
Blonde *bl/bl*	Mich. State U. colony (Pratt and Robbins, 1982)
[2]Brown *b/b*	Huestis stocks (Huestis and Barto, 1934)
Dominant spotting *S/+*	Wild caught in Illinois (Feldman, 1936)
Golden nugget b^{gn}/b^{gn} [in *P. leucopus*]	Wild caught in Massachusetts (Horner and Dawson, 1993)
Gray *g/g*	Natural polymorphism From Dice stocks (Dice, 1933)
Ivory *i/i*	Wild caught in Oregon (Huestis, 1938)
[3]Pink-eyed dilution *p/p*	Sumner's "pallid" deer mice (Sumner, 1917)
Platinum *pt/pt*	Barto stock at U. Mich. (Dodson *et al.*, 1987)
[2]Silver *si/si*	Huestis stock (Huestis and Barto, 1934)
Tan streak *tns/tns*	Clemson U. stock from N.C. (Wang *et al.* 1993)
Variable white *Vw/+*	Michigan State U. colony (Cowling *et al.* 1994)
White-belly non-agouti a^w/a^w	Egoscue's "non-agouti" (Egoscue, 1971)
Wide-band agouti $A^{Nb/a}$	Natural polymorphism. U. Michigan stock (McIntosh, 1954)
Yellow *y/y*	Sumner's original mutant (Sumner, 1917)

MUTATIONS AVAILABLE FROM THE STOCK CENTER[1] (continued)

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)
[4]Boggler *bg/bg*	Blair's *P. m. blandus* stock (Barto, 1955)
Cataract-webbed *cwb/cwb*	From Huestis stocks. (Anderson and Burns, 1979)
[4]Epilepsy *ep/ep*	U. Michigan *artemisiae* stock (Dice, 1935)
[3]Flexed-tail *f/f*	Probably derived from Huestis flexed-tail (Huestis and Barto, 1936)
Hairless-1 *hr-1/hr-1*	Sumner's hairless mutant Sumner (1924)
Hairless-2 *hr-2/hr-2*	Egoscue's hairless mutant (Egoscue, 1962)
[4]Juvenile ataxia *ja/ja*	U. Michigan stock (Van Ooteghem, 1983)

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

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[1]Unless otherwise noted, mutations are in *P. maniculatus*.

[2]Available only as silver/brown double recessive.

[3]Available only as pink-eye dilution/flexed-tail double recessive.

[4]Available from Behavior Mutant Center

Note: Some of the 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 RESOURCES OF THE *PEROMYSCUS* GENETIC STOCK CENTER:

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: Janet Crossland, Colony Manager, Peromyscus Stock Center, (803) 777-3107.

Small numbers (*c.* 5) of deer mice from either of the two distinct inbred lines (H1 and H8) are available from the Stock Center on a limited basis.

Preserved or frozen specimens of types given above.

Tissues, whole blood or serum of types given above.

Flat skins of mutant coat colors or wild-type any of the species above.

Reference library of more than 1900 reprints of research articles and reports on *Peromyscus*.
Copies can be xeroxed and mailed.

Materials are now available through the *Peromyscus* Molecular Bank of the Stock Center. Allow two weeks for delivery. Included is purified DNA or frozen tissues from any of the stocks listed above. Several genomic and cDNA libraries and a variety of molecular probes are available.
(See next page)

PLEASE CALL WITH INQUIRIES.

Peromyscus Genetic Stock Center
University of South Carolina
Columbia SC 29208
(803) 777-3107

Materials on Deposit in the *Peromyscus* Molecular Bank

Accession Number	Item	Description	Species	Donor	Location[1]
Probes and Clones:					
Pr-01	LINE1	pDK62	*P. maniculatus*	D. Kass	C
Pr-02	LINE1	pDK55	*P. maniculatus*	D. Kass	C
Pr-03	ADH1	pADH F72	*P. maniculatus*	M. Felder	B
Pr-04[2]	Mys		*P. leucopus*	(Requested)	
Pr-05[2]	SAT		*P. leucopus*	(Requested)	
Pr-06	6PGD	pB5 clones	*P. californicus*	S. Hoffman	A
Pr-07	MHC *Pele*I	38dp2	*P. leucopus*	M. Crew	A
Pr-08	MHC *Pele*I	52ap6	*P. leucopus*	M. Crew	A
Pr-09	MHC *Pele*I	40Bgl	*P. leucopus*	M. Crew	A
Pr-10	MHC *Pele*I	53Pv1	*P. leucopus*	M. Crew	A
Pr-11	MHC *Pele*I	37B2	*P. leucopus*	M. Crew	A
Pr-12	MHC *Pele*I	37B4	*P. leucopus*	M. Crew	A
Pr-13	MHC *Pele*II	α3E23	*P. leucopus*	M. Crew	A
Pr-14	MHC *Pele*III	17E2	*P. leucopus*	M. Crew	A
Pr-15	MHC *Pema*I	pr44	*P. maniculatus*	M. Crew	A
Libraries:					
Lb-01	lambda genomic	liver (ADH+)	*P. maniculatus*	M. Felder	B
Lb-02	lambda cDNA	liver	*P. maniculatus*	M. Felder	B
Lb-03	lambda genomic	testis	*P. leucopus*	M. Crew	A
Lb-04	cosmid genomic	testis	*P. leucopus*	R. Baker	A
Lb-05	lambda genomic	liver	*P. californicus*	S. Hoffman	A
Frozen Tissue for DNA:					
S-01	bairdii (BW)	liver, other[3]	*P. maniculatus*	Stk. Ctr.	A
S-02	subgriseus (PO)	liver, other	*P. polionotus*	Stk. Ctr.	A
S-03	leucopus (LL)	liver, other	*P. leucopus*	Stk. Ctr.	A
S-04	wild-caught SC	liver, other	*P. gossypinus*		A
S-05	aztecus	liver, other	*P. aztecus*	J. Glendinning	A
S-06	insignis (IS)	liver, other	*P. californicus*	S. Hoffman	A
S-07	inbred PmH1A	liver, other	*P. maniculatus*	Jackson Lab	A
S-08	inbred PmH8	liver, other	*P. maniculatus*	Jackson Lab	A

[1]Location code: **A** = USoCar SAI 01; **B** = USoCar CLS 603; **C** = USoCar CLS 707

[2]Not currently available.

[3]kidney, spleen, testis, carcass.

PEROMYSCUS BEHAVIOR MUTANT CENTER

A Special Stock Center for behavior mutants of deer mice currently is housed at the University of South Carolina-Aiken. The following variants are available from this center.

CONVULSIVE MUTANTS:

Four different convulsive mutants are maintained. Of these four, only two, Chemogenic Convulsive (*CNV*) and Epilepsy (*ep*), have been formally described in the literature.

Alamogordo Convulsive (*ALG*). Affected animals are convulsive after about three months of age and throughout life, with convulsions gradually increasing in severity. In severe seizures, these animals are likely to arch the head and back, to the point of falling over backwards in spasm. This latter behavior is more common in older animals.

Chemogenic Convulsive (*CNV*). Affected animals are convulsive from about one month of age and throughout life, with convulsions gradually increasing inseverity. *CNV/-* mutants tend to display convulsive behavior more readily than *ALG/-* mutants, however the episode is likely to be much less severe.

Epilepsy (*ep*). Convulsions can be elicited in these animals from about twenty-one days of age. These animals usually grow deaf however by about three months of age, and thereafter can no longer be made to convulse. A "waltzing" behavior is often seen in these animals. Differences in the Organ of Corti and the central auditory pathway are associated with this mutation.

Thompson Falls Convulsive (*tf*). Homozygotes convulse throughout life and do not grow deaf. "Waltzing" is not commonly seen. The seizure pattern has a slightly later onset (about three months) and tends to be more severe, sometimes resulting in death.

AGE-DEPENDENT ATAXIAS:

Boggler (*bg*). This is an autosomal recessive mutation characterized by increasing ataxia, tremor, and loss of fine motor coordination. Additional findings suggest that diminished tactile responsiveness also occurs with advancing age. These deficits are correlated with axonal dystrophy and neuronal loss in the CNS.

Juvenile Ataxia (*ja*). This is an autosomal recessive mutation which exhibits a marked ataxia from the time locomotor activity first begins until about forty-five days of age. The phenotype appears to be exaggerated or ameliorated by changes in dietary carbohydrates. Neuronal changes and loss is evident by 120 days of age.

For information about any of these variants, please contact:

Dr. Suellen A. VanOoteghem
Department of Anatomy
School of Medicine
University of West Virginia
Morgantown WV 26506
(304) 284-5443

GENETIC LOCI IN THE DEER MOUSE

(*Peromyscus maniculatus* and allied species)

Table I. lists recognized genetic loci described in *Peromyscus maniculatus* or other species of the *maniculatus*-group. Table II. lists loci formally described in the *P. leucopus* species group, and Table III. those of other species of *Peromyscus*. These lists are limited to loci for which formal Mendelian analysis has been conducted and appropriately reported in the published scientific literature, and/or for which nucleic acid sequences have been published. Additional genetic traits are known some of which have been cited in abstracts, casual reports, newsletters, grant proposals, papers presented at meetings *etc*. The latter are not included, since the descriptions and genetics are generally insufficient to formally define the loci. Presumptive loci described from natural polymorphisms in the absence of formal genetic analysis are not listed here. Protein electrophoretic and other biochemical or immunological variants known in natural populations are listed elsewhere (See **PN #** 15 pp. 17-27 and # 16 pp. 13-27).

Standardization of genetic nomenclature for *Peromyscus* is a function of the Genetic Advisory Committee for the genus. The following guidelines are applied:

1. To the maximum extent feasible, *Peromyscus* genetic nomenclature and conventions will be consistent with those used for other mammalian species, particularly mouse (*Mus*). Where homology is evident or very likely, the same locus name and symbol is employed. Because homology among alleles is more difficult to ascertain, allelic symbols (superscripts) do not necessarily correspond to those of other species.

2. Dominant and incompletely dominant variant or mutant genes are designated with the first letter of the symbol capitalized. Recessive variant or mutant genes are indicated in lower case letters. The wild-type (normal or standard) allele for morphological, pelage color and behavioral traits, when recognized, is symbolized with a "+" sign. Electrophoretic allelic variants of proteins or subunits are indicated by superscripts in alphabetical sequence, except for null alleles which are designated, with an "o" superscript; or, in some cases, by relative mobility with reference to a standard mobility "100". Restriction fragment length variant alleles are are designated by a numerical sequence or size in kilobases. Distinct loci with similar phenotypic effects may be indicated in a hyphenated numerical or alphbetical series.

3. Symbols published by the original investigator are given priority, unless there is clear homology with *Mus* loci, except for certain loci for which the original symbol was retained under the "grandfather" principle and because of prior use in the literature . If an original symbol is in conflict with an established one for *Mus*, the equivalent *Mus* symbol is given preference. In cases where the original symbols have been superseded by subsequent common usage, the latter has been adopted. 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*, priority is given to the first reported, and an alternate designation is devised for the other. (In Table 1 previously published obsolete names and symbols are listed in parentheses.)

4. Presumed loci described solely on the basis of variation observed among individuals in the absence of convincing Mendelian or molecular analysis are not considered to be formally established and are not included in these tables.

5. Linkage assignments are subject to updates of the *Peromyscus* linkage map.

Table 1

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

A. Coat and Eye Pigmentation and Pattern Variants.

Name of locus and allelic variants	Symbol	Mode of inheritance[1]	Linkage group	Definitive description and analysis	Collateral descriptions, interactions and recurrences	Recombination reported
AGOUTI			III			
Wide-band agouti	A^{Nb}	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)		
ASHINESS	*ahy*	recessive		Teed *et al.* (1990)		
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^{ot}	recessive		Egoscue and Day (1958)		
BLONDE[2]	*bln* (*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)	Barto (1942a, 1956), McIntosh (1956a)
PINK-EYED DILUTION	*p*	recessive	I	Sumner (1917) as "pallid"	Clark (1938), Barto (1942b)	Sumner (1922), Clark (1936, 1938), Feldman (1937), Snyder (1960a)
PLATINUM[2]	*plt* (*pt*)	recessive		Dodson *et al.* (1987)		Dodson *et al.* (1987)
RED EYE[2] (Heterochromia)	*rde* (*r*)	recessive		Huestis and Willoughby (1950)		

(Table continued)

Table 1A. Coat and Eye Color Variants (Continued)

Name of locus and allelic variants	Symbol	Mode of inheritance[1]	Linkage group	Definitive description and analysis	Collateral descriptions, interactions and recurrences	Recombination reported
DOMINANT SPOT (Whiteface)	*S*	dominant		Feldman (1936)		Feldman (1937)
SILVER	*si* (*sl*)	recessive	I	Huestis and Barto (1934)		Huestis and Barto (1934), Huestis and Piestrak (1942), Huestis and Lindstedt (1946), Barto (1956)
TAN STREAK	*tns*	recessive		Wang *et al.* (1993)		
VARIABLE WHITE	*Vw*	semi-dominant lethal		Cowling *et al.* (1994)		
WHITE CHEEK[2]	*Wck* (*Wc*)	dominant		Blair (1944)	Bowen and Dawson (1977)	Blair (1944)
WHITESIDE[2]	*ws* (*wh*)	recessive		McIntosh (1956b)		
YELLOWING[2] (Yellow)	*y*	recessive		Sumner (1917)	Sumner and Collins (1922), Clark (1938), McIntosh (1956a)	Sumner (1922), Feldman (1937), Barto (1956), McIntosh (1956a)
Complexly inherited coat pattern traits:						
Minor white spotting (Star, splash, etc.)	*p-1, p-2*	recessive incompletely penetrant		Feldman (1936)	Sumner (1932), Barto and Huestis (1933)	
Grizzled[2]	"*Gr*" (*G*)	"complex dominant"		Sumner (1928, 1932)		
Coat pattern in *P. polionotus*				Bowen and Dawson (1977)	Bowen (1968)	Bowen and Dawson (1977)
Pointed A[2]	*Pt-A* (P_A)	dominant	VII			
Pointed B[2]	*Pt-B* (P_B)	dominant	VII			
Tapered[2]	*Tpt* (*Tp*)	dominant				
Coat pattern modifiers				Bowen and Dawson (1977)		
Squared modifier[2]	*Msq* (*Rs*)	incompletely dominant				
Tapered modifier[2]	*Mtp* (*Rt*)	dominant				

[1]Autosomal unless otherwise stated.

[2]Symbol or name changed to avoid confusion with designations in *Mus*. Obsolete published names and symbols in parentheses.

*No longer known to be in existence

B. Integumentary, Skeletal and Pathological Variants.

Name of locus	Symbol	Mode of inheritance[1]	Linkage group	Definitive description and analysis	Collateral descriptions, interactions and recurrences	Recombination reported
CATARACT-WEBBED[2] (Syndactyly)	*cwb* (*cw*)	recessive		Anderson and Burns (1979)		
FLEXED TAIL	*f*	recessive	I	Huestis and Barto (1936a)		Huestis and Barto (1936a), Huestis and Piestrak (1942), Huestis and Lindstedt (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)	Knapp and Dawson (1991)	
NUDE*[2] (Post-juvenile nude)	*nd* (*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)

[1]Autosomal unless otherwise stated.
[2]Name or symbol changed to avoid confusion with designations in *Mus*. Obsolete published names and symbols in parentheses.
*No longer known to be in existence.

C. Behavior and Neurological Variants.

Name of locus	Symbol	Mode of inheritance[1]	Linkage group	Definitive description and analysis	Collateral descriptions, interactions and recurrences	Recombination reported
BOGGLER[2]	*bgl* (*bg*)	recessive		Barto (1955)	Vandermeer and Barto (1969)	Barto (1955)
EPILEPSY (EP; waltzing in *artemisiae*)	*ep* ("*e*", v_2)	recessive		Dice (1935)	Clark (1938), Watson (1939), Chance and Yaxley (1950), Barto (1954, 1956)	Watson (1939), Barto (1956)
JUVENILE ATAXIA	*ja*	recessive		Van Ooteghem (1983)		
SPINNER*[2] (Waltzing in *rhoadsi*)	*spn* (*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), Dice *et al.* (1963)	Barto (1942a, 1954, 1956), McIntosh (1956a)

[1]Autosomal unless otherwise stated.
[2]Name or symbol changed to avoid confusion with designations in *Mus*. Obsolete published names and symbols in parentheses.
*No longer known to be in existence.

Name of locus	Allelic designation	Linkage group	Definitive description and formal analysis	Recombination reported
ALCOHOL DEHYDROGENASE-1 (liver)	*Adh-1*^{f} *Adh-1*^{s} *Adh-1*^{o}	VI	Felder (1975), Burnett and Felder (1978a, 1978b)	Dawson *et al.* (1983)
ALCOHOL DEHYDROGENASE-2	*Adh-2*		Zheng *et al.* (1993)	
ALBUMIN (serum)	*Alb*^{100} *Alb*^{98} *Alb*^{86}	VI	Brown and Welser (1968), Jensen and Rasmussen (1971)	Dawson (1982), Dawson *et al.* (1983)
AMYLASE (salivary)	*Amy-1*^{a} *Amy-1*^{b} *Amy-1*^{c}	VI	Evans *et al.* (1977)	Dawson *et al.* (1983)
ERYTHROCYTIC ANTIGEN	*Es*^{A} = (*Pm*^{A}) *Es*^{B} = (*Pm*^{B}) *Es*^{C} = (*Pm*^{C})	IV	Rasmussen (1961), Savage and Cameron (1971)	Randerson (1973)
ESTERASE (erythrocytic)[2]	*Es-3*^{o} (*Es-1*) *Es-3*^{a} *Es-3*^{b} etc.	IV	Randerson (1965), Van Deusen and Kaufman (1978)	Randerson (1973)
ESTERASES (tissue and serum)	*Es-1* through *Es-7* (Symbols not standardized)	VIII	Rasmussen and Jensen (1971), Dawson (1982), Gill (1976), Baccus *et al.* (1980)	Dawson (1982)
GLYCEROL-3-PHOSPHATE DEHYDROGENASE[2] (tissue)	*Gdc-1*^{a} (*Gpd-1*) *Gdc-1*^{b}		Gill (1976)	
GLUTAMATE OXALOACETATE TRANSAMINASE (soluble) (ASPARTATE AMINO TRANSFERASE)	*Got-1*^{a} *Got-1*^{b} *Got-1*^{c}		Gill (1976)	Dawson *et al.* (1983)
GLUCOSE-6-PHOSPHATE (AUTOSOMAL HEXOSE-6-P) DEHYDROGENASE[2] (soluble)	*Gpd-1*^{a} (*G6pd-1*) *Gpd-1*^{b}		Shaw and Barto (1965), Shaw (1966)	
HEMOGLOBIN - ALPHA TYPE GLOBINS (Duplicated locus)	*Hba*^{1} = (*Hb*^{f}) = (*Hbl*^{a}) *Hba*^{2} *Hbc*^{o} = (*Hb*^{o}) = (*Hbl*^{o}) *Hbc*^{1} *Hbc*^{2} = (*Hb*^{f})		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} *Hbd*^{o} *Hbb-b1* *Hbd*^{1} or *Hbb-b2* *Hbe*^{o} *Hbb-b3* *Hbe*^{1}	I	Snyder (1978, 1980b), Padgett *et al.* (1967)	Snyder (1980a)
HAPTOGLOBIN (serum)[2]	*Hp*^{1} (*Hpf*) *Hp*^{2}		Rasmussen (1968), Griswold and Dawson (1971)	
IMMUNOGLOBIN (7Sy$_1$)	*Ig*^{f} *Ig*^{s}		Coe (1972)	
LEUCINE AMINOPEPTIDASE (serum)	*Lap-1*^{a} *Lap-1*^{b}	V	Dawson (1982)	Dawson (1982), Dawson *et al.* (1983)
LACTATE DEHYDROGENASE[2] A SUBUNIT (tissue)	*Ldh-1*^{a} (*Ldh-A*) *Ldh-1*^{b}		Cattanach and Perz (1969)	

(Table continued)

Table 1D. Biochemical and Immunological Variants. (Continued)

Name of locus	Allelic designation	Linkage group	Definitive description and formal analysis	Recombination reported
LACTATE DEHYDROGENASE[2] B SUBUNIT (tissue)	*Ldh-2*^{f} (*Ldh-B*) *Ldh-2*^{s}		Shaw and Barto (1963)	
MAJOR HISTOCOMPATIBILITY COMPLEX	*Mhc* (Class I)		Crew *et al.* (1994)	
6-PHOSPHOGLUCONATE DEHYDROGENASE (tissue)	*Pgd-1*^{a} *Pgd-1*^{b}		Gill (1976)	Dawson *et al.* (1983)
PHOSPHOGLUCOMUTASE-1 (tissue)	*Pgm-1*^{a} *Pgm-1*^{b}		Gill (1976)	
PHOSPHOGLUCOMUTASE-4 (tissue)	*Pgm-4*^{a} *Pgm-4*^{b} *Pgm-4*^{c}		Gill (1976)	
SUPEROXIDE DISMUTASE	*Sod-1*^{l} = (*Ng*) *Sod-1*^{p} *Sod-1*^{m}		Birdsall *et al.* (1970)	
TRANSFERRIN (serum)	*Trf*^{a} = (*Trf*^{j}) *Trf*^{b} *Trf*^{c} *Trf*^{e} *Trf*^{m} = (*Trf*^{M})	V	Rasmussen and Koehn (1966), Biggers and Dawson (1971), Griswold and Dawson (1971), Canham *et al.* (1970)	Dawson (1982), Dawson *et al.* (1983)

[1]Autosomal unless otherwise stated.

[2]Symbols changed to avoid confusion with those in laboratory mouse (*Mus*). Obsolete published symbols shown in parentheses.

Table 2

Genetic Loci Formally Described in the *Peromyscus leucopus* Species Group

Name of locus	Symbol and alleles	Mode of inheritance[1]	Reference	Recombination reported
GOLDEN NUGGET	*b*^{gn}	recessive	Horner and Dawson (1993)	
ALBINO	*c*	recessive	Castle (1912)	
CARBONIC ANHYDRASE	*Ca*^{f} *Ca*^{s}	co-dominance	Wilmot and Underhill (1972)	
CATALASE	*Cs*^{a} *Cs*^{b}	co-dominance	Jensen (1969)	
ESTERASE-3 (Esterase-1)[2] (erythrocytic)	*Es-3*^{0} (*Es-1*^{a}) *Es-3*^{b}	semi-dominant	Wilmot and Underhill (1973)	
ESTERASE-2 (serum)	*Es-2*^{0} (*Es-2*^{a}) *Es-2*^{b}	semi-dominant	Wilmot and Underhill (1973)	
HEMOGLOBIN	*Hb*^{A} (in *P. gossypinus*) *Hb*^{B} (in *P. gossypinus*) *Hb*^{C} (in *P. gossypinus*) *Hb*^{D} (in *P. leucopus*)	co-dominance	Foreman (1966)	
MAJOR HISTOCOMPATIBILITY COMPLEX	*Mhc* (Classes I, II; multiple haplotypes)		Crew *et al.* (1989, 1990)	

[1] All are autosomal.

[2]Symbols changed to avoid confusion with those in laboratory mouse (*Mus*). Obsolete published symbols shown in parentheses.

Table 3

Formally Described Genetic Loci in Miscellaneous *Peromyscus* Species

Species	Locus	Symbol and alleles	Mode of inheritance	Reference
P. truei	ESTERASE-1	$Es\text{-}1^{100}$ $Es\text{-}1^{93}$	co-dominance	Zimmerman and Kilpatrick (1975)
P. eremicus	PECTORAL SPOT	*psp*	recessive	Huestis (1925) Clark (1938)
P. californicus	HAIRLESSNESS	*hm*	recessive ?	Packchanian and Louis (1964)

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NOTICE

PEROMYSCUS NEWSLETTER IS NOT A FORMAL SCIENTIFIC PUBLICATION.

Therefore ... *INFORMATION AND DATA IN THE "CONTRIBUTIONS" SECTION SHOULD NOT BE CITED OR USED WITHOUT PERMISSION OF THE CONTRIBUTOR.*

THANK YOU !

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STOWAWAY TRANSPORT RATES OF HOUSE MICE *MUS DOMESTICUS* AND DEERMICE *PEROMYSCUS MANICULATUS*

In memory of J. S. Williams

Stowaway transport rates were obtained from behavioral observations of 14 house mice and 14 deermice during commercial transport. One house mouse escaped during unloading and flaking of 2 500 kg 2-y old oat hay. Three house mice and two deermice were handcaught on trucks during unloading of 51 110 kg transported barley straw and grass hay. One house mouse was handcaught after being buried alive in grain. Two house mice escaped during unloading of a semitruck holding dogfood. House mice (1 live, 6 recently-dead) and deermice (2 recently-dead) were taken from a feedmill screen over which had passed 940 313 kg grain. Ten deermice were trapped in a pickup truck cab. In conclusion, multiplying these transport rates, $7(10^{-5})$ house mice per transported kg hay and $7(10^{-6})$ house mice per transported kg grain, by the worldwide volume of transported hay and grain implies thousands of stowaway house mice occur. Deermice have comparable transport rates.

Experiments (N=82) were done to determine if a particular sex or age predominates among stowaways. For each experiment, I put 1 to 5 hand-caught or trapped house mice in a haybale, let them remain in the haybale for 1 to 13 days, put the haybale in a wheelbarrow, pushed the wheelbarrow for 15 mins, and then searched for transported mice. Stowaways, mice remaining during transport, included 74 of 115 mice (64%), which were primarily lower weight classes, characterizing house mice coming into reproduction. In conclusion, live stowaways should arrive in >50% infested loads.

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Some of our attention still focuses on the major histocompatibility complex (MHC) genes of *Peromyscus*. For example, the hypothesis that MHC genes of a long-living rodent such as *Peromyscus leucopus* might influence the lifespan of a shorter lived rodent such as *Mus musculus* is being tested by transgenic techniques. Transgenic lines harboring six *P. leucopus* MHC (*Pele*) class I genes (out of the forty described in *Immunogenetics* 32:371, 1990) have been derived and are being breed to homozygosity. This may be the first report of transgenic mice derived by the simultaneous microinjection of more than two genes (Crew, Zeller, Walford et al., unpublished). In addition to these transgenic mice, we have described the *P. leucopus* TNFalpha (PeTNF) gene (*Immunogenetics* 35:351, 1991) and now have made transgenic lines which harbor and express this gene. Interestingly, mice expressing the PeTNF transgene exhibit a phenotype which closely resembles ankylosing spondylitis (Crew, Brahn, Effros et al., in preparation). We are hopeful that these mice will provide a valuable model to understand ant treat this form of arthritis.

A *P. maniculatus* cDNA library obtained from the *Peromyscus* Stock Center has been a bonanza. We have cloned and sequenced six unique *P. maniculatus* MHC (*Pema*) class I cDNAs (Crew, Bates, and Douglass, unpublished, but four of the sequences are available on GenBank). Currently we are assessing the evolutionary relationship of *Pema* class I genes to other rodent MHC class I genes using the Molecular Evolutionary Genetic Analysis (*MEGA* v1.01) software developed by M. Nei and colleagues (Penn. St. Univ.). [ADVERTISEMENT: Fax 814-863-7336 or e-mail imeg@psuvm.psu.edu for this powerful and easy to use program]. Moreover, using a novel strategy to screen the *P. maniculatus* cDNA library, we also identified a new MHC class Ib gene termed *PemaT24* and went on to characterize the mouse homolog, *H-2T24* (*Immunogenetics* 39:412, 1994). The *PemaT24* and *H-2T24* genes are now being studied with regards to function.

We hope to develop the MHC of *Peromyscus* species as a paradigm for the evolution of mammalian MHC genes. There is much to do. A genetic map of the *Pele* complex is going to be revealed by pulsed-field gel electrophoresis; the characterization of *mys* insertion sites in and around *Pele* class I genes and class I genes in other *Peromyscus* species will be performed to estimate the rate of evolutionary change; the extent and type of MHC gene polymorphism will be established within and between *Peromyscus* species; and additional *Peromyscus* MHC class Ib genes will be isolated and characterized.

WANTED: COLLABORATORS. Anyone interested in the MHC of *Peromyscus* (for whatever reason) is encouraged to contact us. One notion we kick about relates to the capacity to harbor huge reservoirs of pathogens (e.g. *Borrellia burgdorferi* and hantavirus) that some species of *Peromyscus* seem to possess. Since genes of the MHC are intimately involved in the presentation of foreign antigens to the host immune system it seems reasonable to suspect the MHC genes of *Peromyscus* might somehow relate to host resistance to these pathogens. How can we approach and/or refine this idea in an experimentally tractable manner?

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Hantavirus Studies in Montana

Deaconess Medical Research, Billings Mt., the Indian Health Service, and the Mt. Department of Health has contracted us to perform what medical people call longitudinal studies of hantavirus in deer mouse populations. Basically, they want to know what happens to infection rates in relation to population dynamics.

We have established 18 one ha grids with none east of the continental divide in prairie and shrub step habitats and nine east of the divide in forested and shrub habitats. By the end of September we will have sampled all 18 grids four times. We have collected over 1000 blood samples and there are from 15 to 80 deer mice on various grids. We are using C.J. Krebs' mouse populations analysis package to determine MNAs, survival, recruitment etc.

It is too early for results but we essentially find hantavirus everywhere we look except in high altitude mesic forests occupied by red backed voles and not by deer mice. Other species of voles have antibodies in other habitats. Incidence of the disease in deer mice ranges from 40% of captured animals to 1%. The incidence seems to be declining through the breeding season. We find no indication that there are region-wide population highs or lows of deer mouse as suggested last year in the popular literature. Our bleeding techniques seem to have little or no effect on survival of deer mice.

Among the three of us we have handled tens of thousands of deer mice before this study began. We have been bitten a few times and wore no gloves or respirators as we do now. None of us has antibodies for hantavirus. We now follow rodent handling techniques learned from mammalogists at the Center for Disease Control.

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PEROMYSCUS POPULATION DECLINE: MEASURES OF FLUCTUATING ASYMMETRY

Environmental stress ranges widely from less severe disturbances (e.g., variation in food resource abundance, climatic variation) to extremely harsh disturbances (e.g., habitat destruction due to logging, mining, development) and may contribute to the decline and ultimate extinction of natural populations. Conservation biologists have incorporated measures of fluctuating asymmetry (FA) as indicators of stress in populations. It has been suggested that FA is detectable only in populations experiencing extreme environmental stress (Parsons 1990); however, whether less severe environmental variation negatively affect developmental stability of organisms has not been thoroughly investigated. The objective of this project was to determine if significant levels of FA can be detected during population declines associated with less severe environmental stresses (e.g. periodic food limitations).

From 1990 to 1992, white-footed mice (*Peromyscus leucopus*) populations at Mountain Lake Biological Station (Virginia) declined from 78 mice/ha to 7 mice/ha. Previous work has shown that population fluctuations of *P. leucopus* are directly related to abundance of over-wintering food resources (Wolff, pers. comm.). Loss of over-wintering food resources (acorns) may result in dietary protein deprivation, negatively impacting the health of adults and the growth and development of their offspring. Under these conditions, FA is predicted to increase as food resources become more scarce (Food Abundance Stress Hypothesis). However, as food resources become less abundant, population densities decline. Several researchers (Parsons 1990, Leary et al. 1991, Zakharov et al. 1991) have proposed that levels of FA are directly related to population densities; in other words, FA decreases as population densities decline (Population Density Stress Hypothesis).

Six post-cranial skeletal traits were measured from 239 individuals. The traits examined were lengths of the femur, humerus, ilium, ischium and pubis, and width of the scapula. Five of the traits were measured with hand-held digimatic calipers. The sixth train, scapula width, was measured with the MorphoSys imaging system (Meacham and Duncan, 1989). Relative contribution of measurement error to FA was assessed by repeated measurements of both left and right sides of each skeletal trait. A mixed model, two way ANOVA with replicated measurements of each side (Palmer and Strobeck, 1986; Leamy 1986; Lamb et al., 1990) was used to estimate levels of FA for each skeletal character. Temporal variation in FA measures were tested using a Scheffe-Box homogeneity of variances test.

Significant levels of FA were found in all six bones during the three year duration of the study ($p<.0001$). The femur and humerus, predominant locomotory bones, exhibited less FA than non-locomotory bones. Humerus length was the only trait that showed significant temporal variation in FA ($p<.0001$). FA detected in the humerus decreased as population densities declined, in agreement with the Population Density Stress Hypothesis.

Significant levels of FA were found for all skeletal traits examined during each year of the study. However, the overall results do not support either the Food Abundance Stress Hypothesis or the Population Density Stress Hypothesis. The lack of significant temporal variation in FA measures for most skeletal traits suggests that there may be multiple environmental stresses simultaneously affecting populations during natural density fluctuations, with no single environmental stress overriding all others. This result does not agree with Parson's (1990) prediction that detection of FA requires that populations are exposed to severe environmental stress. Thus, conservation biologists using FA to evaluate the long-term survival of populations should take into account that some measurable level of FA should be expected due to natural variation in environmental conditions.

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Comparison of Predatory Behaviors of Deer Mice

Deer mice and grasshopper mice appear similar morphologically and coexist in many habitats in the western U. S. This study compared certain aspects of their insect-catching behavior in order to get insights into the ecology and evolution of predatory behavior in rodents. The predatory attack of grasshopper mice appears more specialized than that of deer mice: the former attacked faster and more consistently, used their paws to seize a fast moving prey more, showed more resistance to the inhibitory effects caused by novel, aversive or intimidating stimuli associated with a prey than did latter. Nonetheless, deer mice are effective insect predators. In fact, deer mice exhibit a more developed predatory attack than hamsters whose attack response is considered typical of that of many rodents. Grasshopper mice exhibited a number of behavioral and morphological adaptations expected of a mammal that has made the transition to a carnivorous lifestyle and these adaptations also aid in its competition for insect prey with other rodents.
(Details to be published soon in J. Comp. Psychol.)

* * *

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My study of the mice that I have in my colony is primarily one of behavior, especially of mouse-to-mouse reactions in family groups, all female and all male groups, noting cooperation among individuals, cleanliness of groups, eating habits, play periods, sleep periods, and even occasional fighting (the whys when it is obvious). I have discovered a remarkable similarity in behavior of these mice with the behavior of the families of man.

The other part of my study is in recording the color variations in their coats. I still occasionally have a young pup that has a near bald hip area and a bald patch on its head about the time it leaves its nest at weaning time.

The latest variation in color is in the tails of a number of animals. This spring I noted a male with a black tail. He had dark brown on the backs of his hind legs and more dark on his front legs than most of the other mice in the colony. He had been born in a group of females after they had been separated to slow an increase in population. He had not visually developed sexually, and I really did not pay any attention to him until he lost his gray coloration, and developed the unusual dark tail. By that time there were new pups and more on the way.

Many of the young group of mice that I now have after a very prolific breeding season this summer have the very dark tails. It seems this young group are either a very light cinnamon color with tails that look pinkish, and with light brown edges on their ears rather than the near black edges of the ears of the older mice, or else they are almost walnut colored with the near black tails. I do have a few that are a darker cinnamon color with the dark tails. The most unusual that I noted a few days ago are a couple of mice that have the black tops on their tails with black tufts of hair at the ends (other mice showing the tufts had near white tufts). These dark tailed mice do not have the white under the tails, but have sparse hair that is a light charcoal color over a fairly dark gray skin on the tail. Their bellies are a dapple gray rather than white.

Are these normal colorations that I just have not seen in my colony, or are they unusual? I would be interested to know if these same colorations are normal in other colonies. They are new to mine and I have had the colony for over eight years.

* * *

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Possible Circadian Period Mutation in an Albino Deermouse, *Peromyscus maniculatus*.

One albino mutant *P. maniculatus* from the University of South Carolina Peromyscus Stock Center was noted at an age of approximately 2 years to have irregular periods of torpor-like sleep and wakefulness throughout the daily LD 16:8 hr light schedule. Attempts were made to breed the female with a wild type male for genetic analysis of a possible spontaneous circadian period mutation. Since no viable offspring were obtained, continuous recording of her total daily activity pattern was carried out in an LD Schedule of LD 16:8. The mouse was housed inside a constant environment chamber in a transparent 9"x15"x6" plexiglass cage provided with a running wheel and *ad libitum* food and water. Activity was monitored by infrared time-lapse videotaping. For analysis, integer numbers were assigned to each activity type proportional to the intensity of activity, from sleeping through preening, feeding, and wheel running. The average rate of activity per 10-min bin was then calculated and plotted as an actogram for 5 consecutive days. The female displayed sharply delineated periods of activity and rest at approximately 6-hr intervals. Since the abnormal periodicity might have been related to age, gender, or albino condition, activity was also recorded for control groups of *P. maniculatus* including young, wild type males, and old, albino females. These showed normal entrained nocturnal activity rhythms. In addition, a screen of individuals related to the circadian variant female is also currently being carried out. The variant female died at 3 years of age from apparently natural causes.

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HANTAVIRAL INFECTION OF SMALL RODENTS IN NORTH DAKOTA

Two human cases of hantaviral infection have been confirmed in North Dakota since 1992. These, and additional cases from adjacent states, prompted the North Dakota State Department of Health and Consolidated Laboratories and the University of North Dakota to initiate a statewide survey for hantaviral infection in small rodents.

During May-June, 1994, a total of 762 animals representing 18 species of small mammals were live-trapped from 10 sites throughout North Dakota. Laboratory analysis conducted by the North Dakota Department of Health revealed that four species (*Peromyscus leucopus*, *P. maniculatus*, *Microtus ochrogaster*, *M. pennsylvanicus*) carried antibodies for Hantavirus. Of these, 20.1(0.0-40.0)% of 253 *Peromyscus* and 18.2 (0.0-34.4)% of 239 *Microtus* and were seropositive. Tissue samples from the seropositive animals have been sent to CDC to have PCR run to determine strain, and it will probably be some months before we have those results. However, we suspect that the *Microtus* are carrying the Prospect Hill strain (not known to be pathogenic), and the *Peromyscus* are carrying the Sin Nombre (Muerto Canyon) strain. Plans are for continuation of the survey during summer, 1995.

* * *

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