PEROMYSCUS NEWSLETTER

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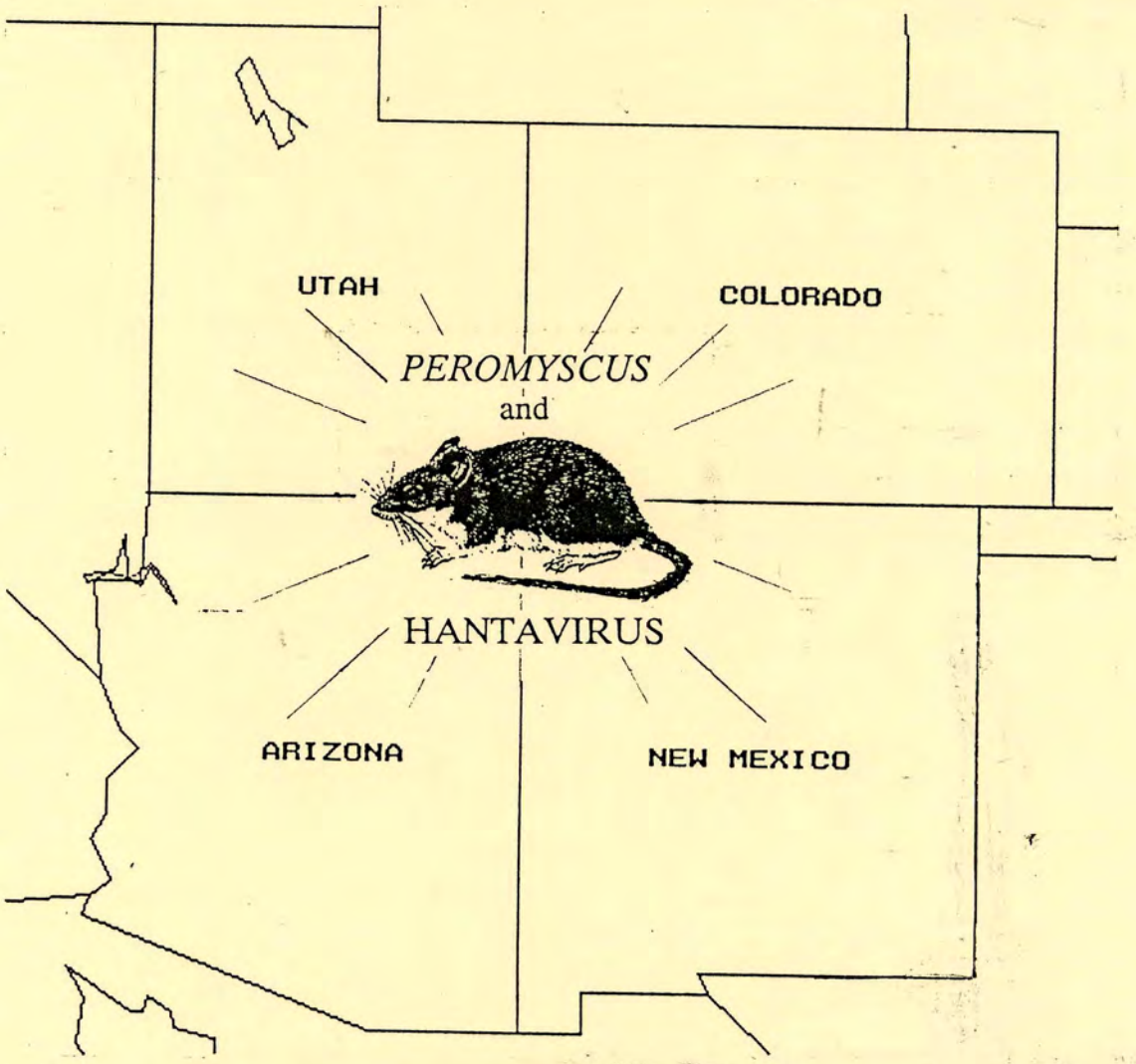

SEPTEMBER 1993

Cover: Four Corners disease and *Peromyscus*
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PEROMYSCUS AND HANTAVIRUS

As many of our readers are now aware, deer mice (*Peromyscus maniculatus*) have been implicated as carriers of the hantavirus which is the likely cause of "Four Corners Disease" or hantaviral pulmonary syndrome (HPS). This mysterious new disease appeared during the spring of this year among residents of New Mexico, Arizona and Colorado. Subsequently other cases were confirmed from Nevada, Texas, Louisiana, California, Oregon, Montana and North Dakota. The disease is characterized by flu-like symptoms, vomiting and high fever. Pulmonary failure and death followed in about two-thirds of the first 38 confirmed cases. A high incidence of the disease occurred on Navajo reservations near Gallup, NM. Most of the affected individuals were active adults aged 20 - 40 years.

The sudden outbreak of this alarming disease, coupled with its high mortality, drew immediate attention from the Center for Disease Control and Prevention (CDC). Plague and Legionnaires' disease were quickly ruled out, but by early June antibody to hantavirus had been detected in 12 patients, and hantavirus-specific RNA sequences were detected in autopsy specimens from several victims.

Hantaviruses, named for the Han-Gang River in South Korea, are RNA viruses (Family **Bunyaviridae**) and are carried by rodents. First recognized in the 1950's as the causative agent of Korean hemorrhagic fever, this disease killed nearly 400 American soldiers during the Korean War. Several additional hantaviruses were subsequently identified in Europe and Asia. Outbreaks of hemorrhagic fever occur periodically in the Orient. In the early 1980's an epidemic near the Chinese-Mongolian border had an exceptionally high mortality.

In Asia the striped field mouse (*Apodemus agrarius*) is the major rodent carrier of the prototype hantavirus (Hantaan virus). However, voles and other rodents also are known to carry other hantaviruses. Laboratory outbreaks of rat-borne (*Rattus norvegicus*) hantaviral infections in Japan and in the former Soviet Union have produced serious illness in technicians. Apparently, cats and dogs rarely carry hantaviruses.

The recent Four Corners hantaviral cases are the first diagnosed in the western hemisphere which could be associated with an indigenous rodent host. The Eurasian hantaviruses produce a disease in which the major effect is renal failure, whereas, the American form is primarily a pulmonary disease. The virus, which has no recognizable effect on the infected rodent, is usually transmitted to humans by inhalation of aerosol particles of rodent urine and feces. Hence, its association with dry conditions or arid habitats. The disease may also be transmitted through rodent saliva. An incubation period of 5 - 42 days, but usually 12 - 16 days, occurs in humans before symptoms appear. Even a brief exposure to the infective particles can result in hemorrhagic fever. Human- to-human tranmission has not been demonstrated.

In CDC tests of wild-caught rodents from the Four Corners region, 31% of 650 deer mice (*P. maniculatus*) exhibited antibody to hantavirus. Furthermore, viral RNA sequences were identified in most seropositive specimens of this species, indicating that the deer mouse may be a chronic carrier. CDC investigators believe **the available information strongly suggests that the deer mouse is the primary reservoir for this newly recognized American hantavirus**. They note that each hantavirus type appears to have a preferred rodent host, but transmission to other species also occurs. In the southwest serologic evidence of possible infection (antibody) has also been found in brush mice (*P. boylii*), pinyon mice (*P. truei*) and western chipmunks (*Tamias* spp). Two of 24 house mice (*Mus musculus*) had high antibody titers. A distinctive viral RNA sequence was identified from a person with acute illness in Louisiana, and cases outside the range of *P. maniculatus*, suggest that more than one variety of hantavirus may be endemic in North America.

Is the hantavirus newly introduced to the U.S. or is it endemic, but previously unrecognized? Although experts differ on this topic, evidence suggests that it has been here all along. The endemic level of the virus may be low under normal population conditions, but rodent populations in the U.S. southwest were as much as 20 times the normal density in 1993 as a consequence of a heavy seed set resulting from excess precipitation the previous year. Increased densities would tend to promote rodent-to-rodent contact and transmission of the virus. Rodents may transmit it among themselves through saliva by biting. Deer mice from locations outside the southwest have tested positive for hantavirus antibody, indicating exposure to the virus. It is also conceivable that there are non-virulent endemic strains in native rodent populations, and virulence in humans results from a viral mutation. Congress has appropriated $ 2.6 million in emergency funding for hantavirus research by CDC, and $ 5.4 million for educational efforts in high risk areas.

An experimental drug, Ribavirin, is effective in treatment of cases of hemorrhagic fever caused by the original (Hantaan) virus, and is available for use in treating patients in the recent American outbreak. Its effectiveness against the Four Corners virus was not clear as of June 1993.

So, for those of us who work regularly with deer mice - **what to do??**

1. Obviously, **caution is in order**, particularly for mammalogists and others collecting wild deer mice and other rodents. **One field biologist, an ornithologist, in California and a rodent trapper in Montana have already died from this disease.** Traps and animals should be handled with rubber or plastic disposable gloves. Avoid collecting in buildings or other enclosures where dust particles may circulate. Use a respirator whenever it becomes necessary handle live or dead wild-caught specimens. Avoid contact with rodent urine or feces. Sanitize traps, cages, dissection instruments and other materials which have been in contact with rodents, preferably with a chlorine bleach solution. Launder any clothing which comes in contact with rodents or their excreta. Consider treating freshly prepared skins and skulls with an intense UV exposure, and skulls or other bones with bleach treatment. Dispose of carcasses in sealed "zip lock" type bags and incinerate. Heed the directions given in **"Hantavirus Infection - Southwestern United States: Interim Recommendations for Risk Reduction"**,

Morbidity and Mortality Weekly Reports (MMWR) 42:RR-11. This report is available in departments of health, medical school libraries and public health schools or by request from CDC, Atlanta, GA 30333. Detailed instructions for those preparing skins and/or skulls, or sampling blood or tissues from wild rodents in high risk areas should contact the **CDC Hantavirus Hotline 1-800-532-9929** for detailed instructions about these or related matters. CDC is willing to test blood from wild-caught rodent specimens. Arrangements can be made by contacting CDC at the hotline number.

2. Individuals who regularly contact wild rodents should have a baseline serum sample stored at - 20° C for reference. If symptoms of a febrile or respiratory illness occur within 45 days of a high risk exposure, immediate medical attention is imperative. The physician should be informed of the patient's possible exposure to hantavirus. A blood sample should be obtained and forwarded by the state health department to CDC for further diagnosis.

3. Established colonies of wild rodent species should be tested for hantavirus antibody. Blood samples from representative animals should be tested by CDC. No wild animals should be introduced into the colony thereafter unless they have been confirmed negative for hantavirus. Wild-caught rodents should be strictly quarantined from laboratory-bred rodents of the same or different species.

4. Mammalogy students and field assistants should be particularly cautioned by teachers and supervisors about hantavirus exposure. All necessary precautions should be exercised. Institutional and personal liability may result from failure to heed preventive measures.

5. Hantavirus may live in cell cultures. Care should be exercised in handling rodent blood and tissues unless they are known to be hantavirus free (See above).

Current Status..... The situation for both field and lab *Peromyscus* researchers is uncertain. At some institutions there has been great concern expressed by animal caretakers and others about safety in handling deer mice and other rodents. We advise those who have contacted the *Peromyscus* Genetic Stock Center **not to panic**, but to exercise the precautions advised above and to follow advice by CDC (See above).

Hopefully, it will become feasible to quickly and inexpensively test for this virus. The high incidence of infection in wild rodents this year well may be an aberration due to high density of the populations, and the proportion of infected animals may decline. Meanwhile, research at CDC, NIH and elsewhere should begin to identify the extent of the problem, details on the mode of transmission among animals, viability of the virus outside the host and other factors relevant to use of *Peromyscus*. We would hope that immunization of animal caretakers and other high risk individuals will soon be feasible. Routine viral screening of laboratory rodents, now required by many institutions, in the future should include hantavirus. While these developments are under way, it is probably wise to avoid unnecessary contact with wild deer mice and other rodents. Consider deferring any collection planned in the near future until the situation becomes clearer.

At the *Peromyscus* Stock Center more than 100 animals representing all of our stocks, as well as all personnel, are in the process of being tested by CDC for hantavirus antibody. Fortunately, thus far, all have tested negative. All animals maintained in the Center are at least two generations removed from the wild. Until the hantavirus-*Peromyscus* situation is better understood, the Stock Center has declared a moratorium on accepting additional stocks. Quarantine and viral screening procedures are being enhanced.

STAY TUNED.

Acknowledgements: Robert Beattie, DVM (Univ. South Carolina Animal Resources), James Childs, ScD (CDC), Francisco Sy, MD, DrPH (Univ. South Carolina School of Public Health), Zongren Wang, MD (Beijing, China, Zoological Institute) and Terry Yates, PhD (Univ. New Mexico) kindly provided information for this report. Information was also obtained from a segment on the "How'd They Do That?" educational series on CBS Television.

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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. Third to sixth generation in captivity.
P. leucopus (LL Stock)	Derived from 38 wild ancestors captured between 1982 and 85 near Linville NC. Seventh to ninth generations in captivity.
P. californicus insignis (IS Stock)	Derived from about 60 ancestors collected between 1979 and 87 in Santa Monica Mts. CA. Fifth to ninth generation in captivity.
P. aztecus	Derived from animals collected on Sierra Chincua, Michoacan, Mexico in 1986 Third to sixth generation 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)
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 colony (Pratt and Robbins, 1982)
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)
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)
Tan streak *tns/tns*	Clemson Univ. stock from N.C. (Wang *et al.* 1993)
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)

MUTATIONS AVAILABLE FROM THE STOCK CENTER* (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)
***Boggler *bg/bg*	Blair's *P. m. blandus* stock (Barto, 1955)
Cataract-webbed *cwb/cwb*	From Huestis stocks. (Anderson and Burns, 1979)
***Epilepsy *ep/ep*	U. Michigan *artemisiae* stock (Dice, 1935)
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)
***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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*Unless otherwise noted, mutations are in *P. maniculatus*.

**Available only on pink-eye dilution background.

***Available from Behavior Mutant Center

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 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 1700 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.

PLEASE CALL WITH INQUIRIES.

Peromyscus Genetic Stock Center
University of South Carolina
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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 finding 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:

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(304) 284-5443

Charles W. Foreman has retired as an active faculty member at the University of the South (Sewanee). Charlie's 1960 report (Am. Mid. Nat. 64:177ff) of hemoglobin polymorphism was the earliest published study using electrophoresis in *Peromyscus*. He subsequently described the inheritance of hemoglobin electromorphs in *P. gossypinus* (1966. Genetics 54:1007ff)

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David Rasmussen of Arizona State University also has retired. David likewise was very active during the earliest days of *Peromyscus* electrophoresis and analyzed the genetics of hemoglobin inheritance in *P. maniculatus* (1968. Biochem. Genet. 2:87ff) and other proteins.

We plan to have a historical account of the early electrophoresis studies in *Peromyscus* in a forthcoming issue of *PN*. Charlie and David figure prominently in this story.

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Twenty-two papers and posters presented at the annual meeting of the American Society of Mammalogists at Bellingham, Washington, in June featured *Peromyscus*.

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Susan E. Smith, 1430 Shalanwood Lane, Placentia, CA 92670, sells attractive T-shirts featuring a Peromyscus grooming its tail. Write her for additional information or to obtain an order form.

* * *

Oops! The Peromyscus Pioneer sketch in *PN* #15 awarded John Christian a non-existent "Ec.D." degree. His degree is an Sc.D. Although Stu Landry authored the account, the mistake was entirely ours, and we apologize.

***

Should the common name for *P. maniculatus* be "deer mouse" as two words or "deermouse" as a single word? It occurs commonly both ways in the literature. F.B. Sumner and Lee Dice hyphenated it as "deer-mouse". The *Peromyscus* Genetic Stock Center Committee recommends two words with no hyphen, "**deer mouse**", as given in the 2nd edition of E.R. Hall's *Mammals of North America.*

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An interesting new report by D.J. Gubernick and J.C. Nordby in Behav. Ecol. Sociobiol. *(1993. 32:211ff) describes mechanisms of sexual fidelity in monogamous* P. californicus. *Some may find it a bit kinky!*

Jim Boone (Univ. Ga.) writes that **Tom Ksiazek** at CDC is interested in obtaining wild rodent blood samples for hantavirus study. Dr. Ksiazek's number is (404) 639-1115. _ _ _ _ _

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HANTAVIRUS HOTLINE: 1-800-532-9929

VARIANT GENETIC LOCI IN NATURAL POPULATIONS OF PEROMYSCUS

Numerous electrophoretic studies of allozymes and other proteins in natural populations of *Peromyscus* have been conducted beginning in the late 1960's. These studies revealed numerous polymorphisms within populations and species, as well as variation among potentially interbreeding species, *e.g. P. maniculatus* and *P. polionotus*. Variants of a protein are generally presumed to identify a genetic "locus", although formal mendelian analysis might not have been accomplished.

PEROMYSCUS NEWSLETTER periodically lists in tabular form the known genetic loci in *Peromyscus* species or species groups. We distinguish between loci which have been formally **demonstrated** and **presumptive** loci. The latter are usually protein variants from natural populations identified by electrophoresis. Separate listings for the two categories are published in PN.

In this issue Tables 1. through 6. summarize presumptive variant loci identified in six species or species groups: *Peromyscus leucopus*, *P. maniculatus*, *P. californicus*, *P. eremicus*, *Podomys (P.) floridanus* and *Megadontomys (P.) thomasi*. Similar tables in PN #15 list variant presumptive loci reported in other *Peromyscus* species and species groups. These tables are updated at three year intervals, thus the next update will be in 1996.

Since limited interbreeding in captivity is frequently possible among different species within a species group, we treat a species group as a single gene pool. Thus, while two species may each be monomorphic for alternate alleles, by hybridization heterozygotes can be produced and genetic analysis conducted. Linkage analysis and gene regulation potentially can be investigated using species hybrids. Such systems are currently used in both *Mus* and *Peromyscus*. Thus, the tables serve as a reference to identify reported variants at given loci. Completely monomorphic loci, *i.e.* loci for which no variation within the species or species group has been reported, are not listed.

Only variants reported in research publications, abstracts excluded, are listed in the tables. References are listed at the foot of each table.

Table 1. VARIANT PROTEIN LOCI REPORTED FROM NATURAL POPULATIONS OF THE *PEROMYSCUS LEUCOPUS* SPECIES GROUP

Protein	Locus	Species	References
Acid phosphatase	*Acp-1*	*P. leucopus*	Nelson *et al.* (1987)
Aconitase	*Acon*	*P. leucopus*	Schnake-Greene *et. al.* (1990)
Adenosine deaminase	*Ada-1*	*P. leucopus*	Krohne and Baccus (1985)
Albumin	*Alb*	*P. leucopus* *P. gossypinus*	Brown and Welser (1968) Jensen and Rasmussen (1971) Browne (1977) Price and Kennedy (1984) Robbins *et al.* (1985)
Alcohol dehydrogenase	*Adh-1*	*P. leucopus*	Robbins *et al.* (1985) Nelson *et al.* (1987) Tolliver *et al.* (1987)
Adenylate kinase	*Ak-1*	*P. leucopus*	Nelson *et al.* (1987)
Amylase	*Amy-1*	*P. leucopus*	Aquadro and Patton (1980)
Carbonic anhydrase	*Ca-1*	*P. leucopus*	Wilmot and Underhill (1972) Krohne and Baccus (1985)
Creatine kinase-1	*Ck-1*	*P. leucopus*	Schnake-Greene *et al.* (1990)
NADH diaphorase	*Dia-1*	*P. leucopus*	Nelson *et al.* (1987)
Esterase	*Es-1* *Es-2* *Es-3* *Es-4* *Es-5* *Es-9*	*P. leucopus* *P. gossypinus*	Price and Kennedy (1980) Wilmot and Underhill (1973) Browne (1977) Smith *et al.* (1984) Robbins *et al.* (1985) Nelson *et al.* (1987) Tolliver *et al.* (1987) Schnake-Greene *et al.* (1990)

Table 1. Variant protein loci in *P. leucopus* group natural populations (Continued)

Protein	Locus	Species	References
Fumarate hydratase	*Fh-2*	*P. leucopus*	Nelson *et al.* (1987)
L-glutamate dehydrogenase	*Gld-1*	*P. leucopus*	Nelson *et al.* (1987)
Glutamate oxaloacetate transaminase	*Got-1* *Got-2*	*P. leucopus*	Price and Kennedy (1980) Nelson *et al.* (1987)
α-Glycerophosphate dehydrogenase	*Gpd-1* *Gpd-2*	*P. leucopus* *P. gossypinus*	Mascarello and Shaw (1973) Browne (1977) Robbins *et al.* (1985)
Glucose-6-phosphate dehydrogenase	*G6pd-1*	*P. leucopus*	Nelson *et al.* (1987)
Glucose phosphate isomerase	*Gpi-1* *(Pgi-1)*	*P. leucopus* *P. gossypinus*	Price and Kennedy (1980) Robbins *et al.* (1985) Nelson *et al.* (1987) Rogers and Engstrom (1992)
Hemoglobin	*Hb*	*P. leucopus* *P. gossypinus*	Foreman (1960) Foreman (1966) Price and Kennedy (1980)
Isocitrate dehydrogenase	*Icd-1* *(Idh-1)* *Icd-2*	*P. gossypinus*	Robbins *et al.* (1985) Nelson *et al.* (1987) Schnake-Green *et al.* (1990)
Lactate dehydrogenase	*Ldh-1*	*P. leucopus*	Robbins *et al.* (1980) Nelson *et al.* (1980)
Malate dehydrogenase-2	*Mdh-2*	*P. leucopus*	Schnake-Greene *et al.* (1990)
Malic enzyme	*Me-1*	*P. leucopus*	Nelson *et al.* (1987) Schnake-Greene *et al.* (1990)

(Continued)

Table 1. Variant protein loci in *P. leucopus* group natural populations (Continued)

Protein	Locus	Species	References
Mannose phosphoisomerase	*Mpi-1*	*P. leucopus*	Rogers and Engstrom (1992)
Major urinary protein	*Mup-1*	*P. leucopus* *P. gossypinus*	Cain *et al.* (1992)
Nucleoside phosphorylase	*Np-1*	*P. gossypinus*	Smith *et al.* (1984) Nelson *et al.* (1987) Schnake-Greene *et al.* (1990)
Peptidase	*Pep-2* (*Pep-B*)	*P. leucopus*	Nelson *et al.* (1987) Schnake-Greene *et al.* (1990)
Phosphogluconate dehydrogenase	*Pgd-1*	*P. leucopus*	Robbins *et al.* (1985) Nelson *et al.* (1987)
Phosphoglucose mutase	*Pgm-1* *Pgm-3*	*P. leucopus* *P. gossypinus*	Mascarello and Shaw (1973) Browne (1977) Price and Kennedy (1980) Robbins *et al.* (1985) Nelson *et al.* (1987)
Sorbitol dehydrogenase	*Sdh-1*	*P. leucopus*	Nelson *et al.* (1987)
Superoxide dismutase	*Sod-1* (*Ipo-1, Tetra-1*) *Sod-2*	*P. leucopus* *P. gossypinus*	Mascarello and Shaw (1973) Browne (1977) Price and Kennedy (1980) Robbins *et al.* (1985) Tolliver *et al.* (1987) Nelson *et al.* (1987)
Transferrin	*Trf*	*P. leucopus* *P. gossypinus*	Price and Kennedy (1980) Robbins *et al.* (1985) Krohne and Baccus (1985)

(Continued)

Table 1. Variant protein loci in *P. leucopus* group natural populations (Continued)

Protein	Locus	Species	References
Xanthine dehydrogenase	*Xdh-1*	*P. leucopus*	Nelson *et al.* (1987)
Non-specific proteins			
Plasma protein	*Ppt-1*	*P. leucopus*	Krohne and Baccus (1985)
General protein	*Gp*		Schnake-Greene *et al.* (1990)

References:

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Table 2. VARIANT PROTEIN LOCI REPORTED FROM NATURAL POPULATIONS OF THE *PEROMYSCUS MANICULATUS* SPECIES GROUP

Protein	Locus	Species[1]	Reference
Acid phosphatase	*Acp-1*	*P. maniculatus*	Baccus and Wolff (1989)
Adenosine deaminase	*Ada-1*	*P. maniculatus*	Baccus and Wolff (1989)
Alcohol dehydrogenase	*Adh-1*	*P. maniculatus* *P. melanotis*	Avise *et al.* (1979) Baccus *et al.* (1980) Massey and Joule (1981) Calhoun *et al.* (1988) Baccus and Wolff (1989)
Albumin	*Alb*	*P. maniculatus* *P. polionotus*	Rasmussen (1970) Jensen and Rasmussen (1971) Selander *et al.* (1971) Avise *et al.* (1974) Biggers and Dawson (1971) Loudenslager (1978) Baccus *et al.* (1980) Calhoun *et al.* (1988)
Aldolase	*Aldo-1*	*P. maniculatus*	Baccus and Wolff (1989)
Amylase	*Amy-1*	*P. maniculatus*	Aquadro and Patton (1980)
Carbonic anhydrase	*Ca-1*	*P. maniculatus*	Baccus and Wolff (1989)
Catalase	*Cat-1*	*P. maniculatus*	Baccus and Wolff (1989)
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) Massey and Joule (1981) Foltz (1981) Aquadro and Kilpatrick (1981) Mewaldt and Jenkins (1986) Baccus and Wolff (1989)

(Continued)

Table 2. Variant protein loci from *P. maniculatus* group populations (Continued).

Protein	Locus	Species[1]	Reference
Glucose dehydrogenase	*Gdh-1*	*P. maniculatus*	Mewaldt and Jenkins (1986) Baccus and Wolff (1989)
Glutamate oxaloacetate transaminase (Aspartate aminotransferase)	*Got-1* *Got-2* (*Aat*)	*P. maniculatus* *P. polionotus* *P. melanotis*	Selander *et al.* (1971) Gill (1976) Loudenslager (1978) Avise *et al.* (1979) Baccus *et al.* (1980) Massey and Joule (1981) Aquadro and Kilpatrick (1981) Calhoun *et al.* (1988) Baccus and Wolff (1989)
Glucose-6-phosphate dehydrogenase	*G6pd-1* (*H6pd-1*)	*P. maniculatus*	Shaw and Barto (1965) Loudenslager (1978) Aquadro and Kilpatrick (1981)
α-Glycerophosphate dehydrogenase	*Gpd-1*	*P. maniculatus* *P. polionotus* *P. oreas*	Selander *et al.* (1971) Mascarello and Shaw (1973) Gill (1976) Avise *et al.* (1979) Calhoun *et al.* (1988) Baccus and Wolff (1989)
Glucose phosphate isomerase	*Gpi-1* (*Pgi-1*)	*P. polionotus* *P. melanotis* *P. maniculatus*	Selander *et al.* (1971) Avise *et al.* (1974) Avise *et al.* (1979) Massey and Joule (1981) Foltz (1981) Baccus and Wolff (1989)
Glutamate pyruvate transaminase	*Gpt-1*	*P. maniculatus*	Baccus and Wolff (1989)

(Continued)

Table 2. Variant protein loci from *P. maniculatus* group populations (Continued).

Protein	Locus	Species[1]	Reference
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 (1978, 1980) Loudenslager (1978) Avise *et al.* (1979) Massey and Joule (1981) Aquadro and Kilpatrick (1981) Chappell and Snyder (1984)
Haptoglobin	*Hpt*	*P. polionotus*	Peck and Biggers (1975)
Immunoglobin (7Sγ)	*IgG*	*P. maniculatus*	Coe (1972)
Isocitrate dehydrogenase	*Idh-1* *(Icd-1)*	*P. maniculatus* *P. oreas* *P. polionotus* *P. sejugis*	Mascarello and Shaw (1973) Baccus *et al.* (1980) Avise *et al.* (1974) Massey and Joule (1981) Aquadro and Kilpatrick (1981) Calhoun *et al.* (1988) Baccus and Wolff (1989)
Lactate dehydrogenase	*Ldh-1* *Ldh-2*	*P. maniculatus* *P. polionotus* *P. melanotis*	Selander *et al.* (1971) Avise *et al.* (1979) Massey and Joule (1981) Mewaldt and Jenkins (1986) Calhoun *et al.* (1988)
Malate dehydrogenase	*Mdh-1* *Mdh-2*	*P. maniculatus* *P. polionotus*	Selander *et al.* (1971) Massey and Joule (1981)
Malic enzyme	*Me-1*	*P. maniculatus*	Baccus and Wolff (1989)
Nucleoside phosphorylase	*Np-1*	*P. maniculatus*	Baccus and Wolff (1989)

(Continued)

Table 2. Variant protein loci from *P. maniculatus* group populations (Continued).

Protein	Locus	Species[1]	Reference
Peptidase	*Pep-1* *(Pep-B)* *Pep-2*	*P. maniculatus* *P. melanotis*	Avise *et al.* (1979) Baccus *et al.* (1980) Massey and Joule (1981) Calhoun *et al.* (1988) Baccus and Wolff (1989)
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) Massey and Joule (1981) Foltz (1981) Mewaldt and Jenkins (1986) Baccus and Wolff (1989)
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) Massey and Joule (1981) Aquadro and Kilpatrick (1981) Baccus and Wolff (1989)
Sorbitol dehydrogenase	*Sdh-1*	*P. maniculatus*	Baccus *et al.* (1980) Massey and Joule (1981)
Superoxide dismutase	*Sod-1*	*P. maniculatus*	Baccus and Wolff (1989)
Transferrin	*Trf*	*P. maniculatus* *P. polionotus*	Rasmussen (1970) Biggers and Dawson (1971) Selander *et al.* (1971) Avise *et al.* (1974) Gill (1976) Redfield (1976) Loudenslager (1978) Avise *et al.* (1979) Baccus *et al.* (1980) Massey and Joule (1981) Foltz (1981)

(Continued)

Table 2. Variant protein loci from *P. maniculatus* group populations (Continued).

Protein	Locus	Species[1]	Reference
Miscellaneous non-specific proteins (pre- and postalbumins *etc.*)		*P. maniculatus*	Mascarello and Shaw (1973) Gill (1976) Baccus and Wolff (1989)

[1]Species from which protein variants were obtained.

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Table 3. VARIANT PROTEIN LOCI REPORTED
FROM NATURAL POPULATIONS OF *PEROMYSCUS CALIFORNICUS*

Protein	Locus	References
Albumin	*Alb*	Avise *et al.* (1974)
Esterase	*Es-3* *Es-4* *Es-4+* *Es-5*	Smith (1979)
α-glycerophosphate dehydrogenase	*Gpd-1*	Avise *et al.* (1974) Smith (1979)
Isocitrate dehydrogenase	*Idh-1* *Idh-2*	Avise *et al.* (1974) Smith (1979)
Malate dehydrogenase	*Mdh-1*	Avise *et al.* (1974) Smith (1979)
Malic enzyme	*Me-1* *Me-2*	Smith (1979)
Post-albumin	*Palb*	Smith (1979)
Peptidase	*Pep-1*	Smith (1979)
6-Phosphogluconate dehydrogenase	*Pgd-1*	Avise *et al.* (1974) Smith (1979)
Phosphoglucoisomerase	*Pgi-1*	Smith (1979)
Phosphoglucomutase	*Pgm-1* *Pgm-3*	Avise *et al.* (1974) Smith (1979)

(Continued)

Table 3. Protein variants in *P. californicus* natural populations (Continued)

Protein	Locus	References
Sorbitol dehydrogenase	*Sdh-1*	Avise *et al.* (1974) Smith (1979)
Transferrin	*Trf*	Avise *et al.* (1974)

References:

Avise, J.C., M.H. Smith, R.K. Selander, T.E. Lawlor and P.R. Ramsey. 1974. Syst. Zool. 23:226-238.
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Table 4. VARIANT PROTEIN LOCI REPORTED FROM NATURAL POPULATIONS OF *PEROMYSCUS EREMICUS* AND RELATED SPECIES

Protein	Locus	Species	References
Alcohol dehydrogenase	*Adh-1*	*P. eremicus*	Avise *et al.* (1974)
Amylase	*Amy-1*	*P. eremicus*	Werbitsky and Kilpatrick (1987)
Esterase	*Es-1*	*P. eremicus*	Rasmussen and Jensen (1971) Avise *et al.* (1974)
Glutamate oxaloacetate transaminanse	*Got-1*	*P. eremicus*	Avise *et al.* (1974)
α-Glycerophosphate dehydrogenase	*Gpd-1*	*P. eremicus*	Avise *et al.* (1974)
Isocitrate dehydrogenase	*Idh-1* *Idh-2*	*P. eremicus* *P. guardia* *P. interparietalis*	Avise *et al.* (1974)
Lactate dehydrogenase	*Ldh-1*	*P. eremicus* *P. caniceps*	Avise *et al.* (1974)
Phosphogluconate dehydrogenase	*Pgd-1*	*P. eremicus* *P. caniceps*	Avise *et al.* (1974)
Phosphoglucomutase	*Pgm-1*	*P. eremicus*	Avise *et al.* (1974)
Plasma protein B (Macroglobulin)	*Ppb*	*P. eremicus* *P. caniceps*	Avise *et al.* (1974)
Transferrin	*Trf*	*P. eremicus* *P. merriami* *P. caniceps*	Rasmussen and Koehn (1966) Avise *et al.* (1974)

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Table 5. VARIANT PROTEIN LOCI REPORTED FROM NATURAL POPULATIONS OF *PEROMYSCUS (PODOMYS) FLORIDANUS*

Protein	Locus	Reference
Esterase	*Es-1* *Es-2* *Es-4*	Smith *et al.* (1973)
Glutamate oxaloacetate transaminase	*Got-1*	Smith *et al.* (1973)
Hexose-6-phospahte dehydrogenase	*Gpd-1*	Smith *et al.* (1973)
Hemoglobin	*Hb-1*	Smith *et al.* (1973)
Isocitrate dehydrogenase	*Idh-1* (*Icd-1*)	Smith *et al.* (1973) Rogers and Engstrom (1992)
Lactate dehydrogenase	*Ldh-1* *Ldh-2* *Ldh-3*	Smith *et al.* (1973) Rogers and Engstrom (1992)
Malic enzyme	*Mod-1*	Smith *et al.* (1973)
Phosphoglucomutase	*Pgm-1* *Pgm-3*	Smith *et al.* (1973)
Pre-albumin	*Pra*	Smith *et al.* (1973)
Transferrin	*Trf*	Smith *et al.* (1973)

Reference:

Rogers, D.S. and M.D. Engstrom. 1992. J. Mamm. 73:55-69.
Smith, M.H., R.K. Selander and W.E. Johnson. 1973. J. Mamm. 54:1-13.

Table 6. VARIANT PROTEIN LOCI REPORTED FROM NATURAL POPULATIONS OF *PEROMYSCUS (MEGADONTOMYS) THOMASI*

Protein	Locus	References
Alcohol dehydrogenase	*Adh-1*	Werbitsky and Kilpatrick (1987)
Albumin	*Alb*	Werbitsky and Kilpatrick (1987)
Amylase	*Amy-1*	Werbitsky and Kilpatrick (1987)
Carbonic anhydrase	*Car-1*	Werbitsky and Kilpatrick (1987)
Cholinesterase	*E-2*	Werbitsky and Kilpatrick (1987)
Glutamate oxaloacetate transaminase	*Got-1*	Werbitsky and Kilpatrick (1987)
Hemoglobin	*Hba-1*	Werbitsky and Kilpatrick (1987)
Phosphoglucoisomerase	*Pgi-1*	Werbitsky and Kilpatrick (1987)
Peptidase	*Pep-1* (*Pep-A*) *Pep-4* (*Pep-D*) *Pep-B1*	Werbitsky and Kilpatrick (1987) Rogers and Engstrom (1992)
Transferrin	*Trf*	Werbitsky and Kilpatrick (1987)

Reference:

Rogers, D.S. and M.D. Engstrom. 1992. J. Mamm. 73:55-69.
Werbitsky, D. and C.W. Kilpatrick. 1987. J. Mamm. 68:305-312.

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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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Studies of the Cotton Mouse (*Peromyscus gossypinus*)

Our studies of the distribution of genetic and morphological variation in small intervals of time and space are yielding results. While we have finished the data collection and lab work, we have yet to fully analyze the data. However, we have made some interesting and unexpected observations of the data.

In the genetic data, for example, we have two samples taken from the same place in February 1991 (n = 25) and August 1993 (n = 22), and a third sample taken in August 1993 (n = 22) from a site 5-8 km downstream from the first site. At the Transferrin locus, the 1991 sample has 2 alleles with frequencies of 0.78 and 0.22, and the 1993 sample at the same site had the same 2 alleles with frequencies of 0.98 and 0.02. At another locus, Carbonic Anhydrase-1 differs between the two sites where samples were taken at the same time. They both have the same common allele, but at one site the frequencies of two uncommon alleles are 0.14 and 0.02, whereas at the other site they are basically reversed: 0.02 and 0.09. Frequencies of alleles at this locus are approximately identical in the temporal samples. We have many examples of these small-scale temporal and geographic differences.

The morphological data also shows differences among groups that we would not expect to be different. For example, we have found significant differences among males and females where sample sizes are large (accounting for age differences). However, we found that the characters where the sexes differ are not the same from site to site.

We are also continuing to work on the distribution of Lyme Disease in the Southeast and have recently made contract with the CDC concerning the Hantavirus that has killed several people in the Desert Southwest and more recently killed people in Texas and Louisiana. We will be collaborating with the CDC by providing tissue samples. Tom Ksiazek at the CDC is interested in contacting anyone collecting rodent blood samples. Call Ksiazek at (404) 639-1115 for more information.

* * *

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[*Addenum to entry in Peromyscus Newsletter # 15*]

More information regarding "black" Peromyscus has come to light. J. Bristol Foster's Ph.D. thesis on the mammals of the Queen Charlotte Islands mentions "the occasional melanistic forms found on Frederick Island." [This information was reeceived after PN # 15 was in press.]

* * *

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Catechol Differences in the Brains of Two Color Morphs of *Peromyscus*

Levels of norepinephrine (NE) and dopamine (DA) from the cortex, striatum, and brain stem were measured by HPLC from brains of 6 agouti and 6 extreme non-agouti (black) deermice (*Peromyscus maniculatus gracilis*). The pattern of NE and DA was statistically and strikingly different in the two color morphs. Levels of NE were statistically similar in the two coat-color morphs in the cortex and striatum although NE in non-agouti animals was 8% higher and less variable in both regions. However, NE in the brain stem was significantly higher (5.2 vs 4.3 nmoles/g wet weight) in agouti deermice with low and similar variability in both morphs. The pattern was the opposite for DA. Agouti animals had nearly twice the DA in both the cortex and the striatum although the high variability prevented statistical significance. In contrast, brain stem levels of DA were statistically higher (.35 vs .25 nmoles/g wet weight) in non-agouti animals and variability was much lower. These two color-morphs of deermice may provide an excellent and natural model system for investigating catechol related physiological or behavioral processes.

This work has been submitted to Comparative Biochemistry & Physiology C.

* * *

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More Mutants

My colony of *Peromyscus* continue to produce a variety of coat colorations and markings. Recently one group produced two very pale gray males. The oldest of the gray males is now beginning to get brown patches on his coat. At first he had a cinnamon brown marking on the end of his nose and between his ears. About six weeks or so ago he began to show patches of the brown along the lower edges of his hips. This slowly has moved up the sides of his hips so that he is brown about half way up his sides. About 4 or 5 days ago he developed a brown patch on his shoulders that looks almost like a saddle. I suspect he may eventually turn all brown, but is an unusually marked animal right now. Two nights ago the younger one died.

This group is very prolific. There are several bald pups in the cage. and one that has a bare back as well as a bald head. One of the nursing females has deformed front feet that turn inward. The toes do not seem to spread and she walks on the sides of the feet that appear to be swollen. She is able to pick up food, but holds it more in a fist position than in an open hand position. She grooms her face rapidly before eating and has raised many pups, so seems to have no distress from the deformity.

This group started from five adult mice about a year ago. I have removed 20 mice to other cages and need to remove many more again.

A couple of months ago I removed seven mice. One female from these seven gave birth to five pups that evening. She did not go to the pups after their birth but went to a jar with the other six mice. The next morning one of the pups was dead and the four remaining looked like they had never been fed. At least one of the females in the original cage was nursing, so I decided to see if these could be saved by one of them. I saw a female drag three or four pups on nipples into a nest jar so I put one of the abandoned pups by the jar entrance. Almost immediately a head reached out, grabbed the pup and pulled it inside the jar. I put a second one outside the jar with the same result. She pulled the last two inside with her also.

Because she and/or other females took care of her pups and the abandoned pups I do not know which of the pups (now weaned) are hers and which one are those abandoned pups, but they all survived. It is among this group that the bald-headed pups and the one with no hair on its back, have appeared. They seem a bit smaller than the pups with the normal gray coats, so may be the abandoned pups that were never fed for at least 15 or more hours. Then again they may be smaller just because they are a form of mutant. They are active and appear to be healthy.

Another possibility as to the cause of their smaller size may be the number of pups that mother was feeding, and they were a bit younger than her own pups, so perhaps they did not get the best "feeding stations". I do think that more than one female nursed them. There seemed to be three females that dragged pups around on their nipples. One could have been the pups' mother that I put back in that cage after the pups were safely tucked away inside the nursery jar.

(Continued)

Documentation

I am still documenting as much of my study as possible on film and on camcorder tapes. I do have photos of these unusual coat markings or lack of coats, and am taking new photos of the individual mice at intervals that show what changes develop. The camcorder is great to show behavior that is impossible to show in stills. It records dexterity, agility, grooming, eating habits, family relationships, and sexual feats that no one would believe without observing such. These recordings give proof of the anecdotal stories about these interesting animals. I urge any of you who are doing behavioral studies to consider using this wonderful tool.

Individual Recognition

In June I removed a male from his two male cagemates to have a tumor removed from his nose. Unfortunately he bled so much the veterinarian had to stop the surgery before he had completely removed the tumor. He feared the animal would bleed to death. He closed the incision with a special glue which held fine. Unfortunately the animal has had trouble breathing normally since then. At first his breathing was quite noisy. Now it is quiet but he never closes his mouth completely. He has a good appetite but is not as active as he used to be. He was a backwards flipper. He no longer does that. I think the growth may be getting larger so his days may be numbered. I decided to put him back with his cagemates since these animals seem to need company. I wondered how this would work after his being alone for three months. Also since they are all males, and males usually fight if not together since very young, I decided putting him back would be risky, but worth it if there is recognition among them.

I put him in the cage and the other two came forward immediately. They both greeted him and the three went in the jar cuddling and grooming one another. There was not even the submissive stance when males don't want to fight. It was as if he had never been away. Certainly this proves that these mice have a memory of family that lasts at least three months. They are still together. It is nearly two weeks now. They still sleep cuddled together and I have seen no signs of any battling among them.

I have put some males together that would have battled to the death I believe. I have put some together that after a day or so of tiffs and submissive stances, have settled down and gotten in a jar together. As long as there are no females near the cages they learned to live in harmony. But this is the first time males have shown genuine affection immediately definite recognition.

* * *

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EFFECTS OF CONSUMPTION OF FUNGALLY-INFECTED TALL FESCUE SEEDS ON *PEROMYSCUS*

Many grasses, including several agronomically important forage species, harbor endophytic, ascomycetous fungi, such as *Acremonium coenophialum*. These fungal endosymbionts markedly affect the characteristics of infected (E+) grass individuals, compared with uninfected (E-) conspecifics, influencing the physiology, morphology, reproductive biology and palatability of grass hosts. One characteristic seen in E+ host plants is the presence of fungally produced alkaloids, which, if consumed by vertebrates, may produce central nervous system (CNS) effects. In addition to CNS effects, the ingestion of E+ forage by cattle, sheep, and horses has detrimental effects on locomotion, the cardiovascular system, thermoregulation, food consumption, growth, and reproductive performance. Some of these effects, however, are not manifested by all of these species, suggesting the existence of species-specific target tissues as well as general effects attributable to E+ ingestion. A paucity of information exists regarding the effects of ingested E+ seeds and other plant parts on rodents; those data that are available are derived from studies of domestic (white) rats and mice.

During the past two years, we have been supported by the NSF-REU program as we seek to determine the systemic effects of consumption of E+ tall fescue (*Festuca arundinacea*) seeds by a native rodent granivore, the white-footed mouse. Knowledge of the adverse effects of consumption of E+ seeds is an important step in documenting the selective pressures favoring mouse avoidance of these seeds.

In 1992, Sara Seematter, Dana Zimmerman, and I fed breeding pairs and individual white-footed mice various combinations of chow, infected seeds, and uninfected seeds. As we expected, consumption of diets rich in seeds of either type severely depressed reproductive output of the pairs and retarded growth of the reproductive tract in individual males and females. Fungally-infected seeds, however, did not suppress reproduction any more than did non-infected seeds. These results are surprising considering the well-documented effects of these diets in lab rodents. We also found no effect of diet on gastrointestinal morphology despite the higher fiber content of the seed vs. chow diets.

In 1993, Jamie Barger has attempted to construct water and sodium budgets for both male and female *P. leucopus* consuming chow diets or those composed primarily of infected or uninfected tall fescue seeds. Most seeds have very low sodium content, such that mice consuming them tend to compensate for reduced sodium intake by reducing urinary losses. Extracts from infected seeds, however, have been shown by other labs to inhibit the activity of Na/K-ATPase, which may jeopardize the ability of consumers of infected seeds to resorb sodium from their filtrate. Preliminary results do not indicate substantial differences between mice eating the two types of seeds; data are still being collected as samples are being processed this fall.

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