



Avian Genetic Resources at Risk:

An Assessment and Proposal for Conservation
of Genetic Stocks in the USA and Canada



Clockwise from above:

- White Leghorn rooster from UCD 003, a highly inbred stock that serves as genetic background to a number of congenic chicken stocks.
- Red Jungle Fowl rooster from UCD 001, a highly inbred stock derived from the wild chicken progenitor species.
- Hybrid rooster from cross between Red Jungle Fowl (UCD 001) and White Leghorn (UCD 003). Offspring from the cross of this rooster and a White Leghorn hen were used as a reference population in developing the chicken genome map.

(Photographs courtesy of J. Clark, University of California–Davis.)

Front cover: Chicken, turkey, and quail eggs.
(Photograph courtesy of J.M. Pisenti, University of California–Davis)

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Preface

IN 1984, AN INTERNATIONAL SYMPOSIUM and Workshop on Genetic Resources Conservation for California made the recommendations that precipitated the establishment of the Genetic Resources Conservation Program within the University of California (UC/GRCP). These recommendations recognized that the avian genetic stocks developed in California, primarily chicken and coturnix quail, had been valuable in research and the commercial poultry industry and that continued conservation of such stocks depended upon long-term support for live-bird maintenance and further research on cryopreservation technology. At that time, there was no national-level program, comparable to the National Plant Germplasm System, directed to animal genetic resources. By the early 1990s, the California situation was fast approaching a crisis with the imminent retirement of the two primary developers and curators of poultry research genetic stocks at the University of California—Davis, Ursula Abbott and Hans Abplanalp. No plan was in place for the continued support and maintenance of the important and widely used genetic stocks developed and acquired by them. Nationally, other collections were at risk as the poultry and avian science departments in the US were losing faculty (to retirements) and financial resources that had supported collection maintenance in the past. The UC Davis campus recently followed this trend, with the merging of its Avian Sciences Department into the Animal Science Department in 1997. Avian genetic stocks in Canada are likewise imperiled.

Recognizing the drastic reductions in human and financial resources for avian genetic resources management in the US and Canada, UC/GRCP, in cooperation with two agencies of the US Dept. of Agriculture (USDA), the Agricultural Research Service (ARS) and the Cooperative State Research, Education, and Extension Ser-

vice (CSREES), convened a binational Task Force in 1995 to address the problem. The Task Force included representatives from the many public institutions that have or had poultry research programs, from private companies, and from research programs that have utilized poultry genetic stocks.

In support of the Task Force's work, UC/GRCP conducted a survey of existing genetic stocks in the US and Canada to provide an inventory for analysis. This new inventory is presented in this document. The most recent prior inventory was published in 1988 (R.G. Somes, Jr. 1988. *International Registry of Poultry Genetic Stocks*. Storrs Agr. Exp. Sta. Bull. 476. The University of Connecticut, Storrs). It was not surprising to find that many stocks had been lost in the interval. Indeed, numerous stocks have been lost over the course of this Task Force effort.

The majority of the Task Force convened at an initial meeting in Davis in October 1995 and a subgroup met in Washington DC in May 1996. Results of the survey and progress of the Task Force have been presented and discussed at several meetings including the annual NE-60 Regional Research Project meetings and the annual meetings of the Poultry Science Association and the Society for Developmental Biology. An interim review of the issues was presented by M.E. Delany, co-chair of the Task Force, and J.M. Pisenti, UC/GRCP's facilitator for the Task Force (M.E. Delany and J.M. Pisenti. 1998. Conservation of poultry genetic research resources: Consideration of the past, present, and future. *Poultry and Avian Biol. Rev.* **9**:25–42).

We thank Mary Delany (University of California–Davis) and Robert L. Taylor, Jr. (University of New Hampshire) for serving as co-chairs of the Task Force and all the members for their contributions of information and editing of this report. We especially acknowledge Jacqueline Pisenti, a

poultry developmental geneticist, who facilitated this effort in every way. Her great familiarity with poultry stocks and expertise in maintaining the genetic stocks at UC Davis was a definite asset in carrying out the charge to the Task Force. Her persistence in pursuit of information for the survey made it a successful venture. Her work in pulling together this report from components submitted by many different individuals has produced what we think is a document that speaks in a uniform, strong voice for the value of these genetic stocks and for the imperative of their continued conservation.

The work of the Task Force could not have been accomplished without financial support from UC/GRCP and from grants from the National Science Foundation and the Agricultural Research Service. These grants both supported the activities of the Task Force and contributed to the maintenance of at-risk poultry genetic resource collections at the University of California-Davis.

The Task Force has defined the types of genetic stocks, made an excellent summary of the extent of the use of avian genetic resources in many different research disciplines, analyzed the inventory for trends in stock development and conservation, and finally made recommendations designed to ensure the continued availabil-

ity of existing genetic resources and support the further development of new genetic resources.

While Congress authorized the US National Genetic Resources Program with the Food, Agriculture, Conservation and Trade Act of 1990, there still is no plan for ensuring the conservation of poultry and other livestock genetic resources critical to the US. In Canada there has been a loss of federal support for poultry genetic resources and now most of their important genetic resources exist only as frozen embryo blastodisc cells. With these uncertainties, it is doubtful if new genetic stocks from current research will be conserved. This is a direct deterrent to new research on critical issues in avian biology. At the same time, we recognize that emerging technologies of molecular biology and genomics have increased the value of existing genetic stocks and provided the impetus to develop new ones which will need conservation attention. The Task Force has made well considered and reasonable recommendations. UC/GRCP and the USDA will work to make these recommendations known and will support their adoption into a viable Avian Genetic Resources System to protect and make available to all researchers extant avian genetic stocks and, importantly, to encourage new research in biomedicine, biology, and agriculture.

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Executive Summary

Background

AMONG THE DOMESTICATED AVIAN species, traditional breeds developed for meat or egg production are a tremendous source of genetic diversity. Such breeds result from hundreds of years of selection by farmers and breeders, incorporating a variety of unique genes and gene combinations. Likewise, intense selection for odd and interesting traits resulted in the array of fancy breeds treasured by hobbyists. The relatively small size of most poultry species (particularly the chicken and Japanese quail), along with their phenomenal reproductive capacity and genetic variability, make them particularly well suited to multi-generational studies with rigorous selection or high levels of inbreeding. This has stimulated the development of many important genetic stocks of chicken, turkey, and quail by researchers in agricultural departments at public universities or in federal agricultural research organizations. These specialized genetic stocks have contributed widely to research in basic, biomedical, and agricultural sciences.

To date, no formal conservation plan exists for these stocks at any organizational level. Instead, conservation of these important avian genetic resources is largely dependent upon individuals, sometimes connected through breed associations or academic circles, but largely as independent efforts. This lack of a formal conservation plan is particularly perilous for research genetic stocks. Unlike the more widely dispersed traditional or hobby breeds, specialized research stocks are often kept at only one location, which makes them far more vulnerable to extinction. In particular, when such stocks can no longer be maintained by their curating researcher, they are often perceived as an unnecessary drain on dwindling financial resources at that institution, and may be eliminated at very short notice.

Findings

This study documented the loss of more than 200 stocks since 1984. A detailed database was prepared for all extant stocks in the US and Canada, including 268 chicken, 20 turkey, 65 Japanese quail, and 8 waterfowl or gamebird accessions.

1. The continuing loss of avian research stocks is a significant concern, as in the past 15 years more than 200 mutant, inbred, and selected avian genetic stocks were destroyed. More than one-third of the remaining stocks are at risk of elimination within the next few years.
2. While several research institutions in the US and Canada still maintain collections, they are at capacity and will likely face reduction in resources in the near future. There is no facility currently available that can accommodate the needs of all currently at-risk stocks.
3. Without stock conservation facilities, there will be little incentive for investment in lines of research that would produce new genetic stocks, particularly highly inbred and selected stocks. This is a direct deterrent to research on critical issues in avian biology.
4. Many research stocks have unique genetic properties which would be impossible or very costly to recreate. Some of these have high potential value for future genetic research in the basic, biomedical, and agricultural sciences, for example:
 - Uniform selected and inbred stocks are genetically well suited for studies of the

- immune system and other complex functional systems.
- Strains that carry a wide variety of mutations affecting specific developmental or metabolic processes provide a starting point for research exploring the tissue or cellular source of defects in embryonic development and metabolic function, leading to a better understanding of the control of the normal processes.
 - Mutations that closely mimic certain human congenital defects or disorders provide animal models for detailed experimental analysis of such conditions.
5. Information regarding the properties and availability of research stocks is not readily accessible to researchers who could effectively use them.
 6. Many researchers who utilize genetic stocks, especially in basic biological and medical research areas, do not have facilities for maintaining stocks and require consistent and reliable sources for live birds, tissues, or eggs.
 7. Avian genetic stocks can be conserved as live animals or cryopreserved germplasm (specifically as embryonic cells, semen, or embryos).
 - Live bird maintenance is expensive and labor intensive, as many research stocks require rigorous disease control measures with specialized rearing and adult bird housing conditions, yearly monitoring of specific performance traits for selected stocks, and genetic testing for chromosomal abnormality and mutant-carrier stocks.
 - Although the annual storage cost for cryopreserved germplasm is relatively low, very little germplasm from avian genetic stocks is currently cryopreserved, and recovery of live birds from the cryopreserved semen can be quite difficult. An additional disadvantage of semen cryopreservation is that only the male genome is conserved. This eliminates or severely limits the usefulness of this conservation technique with inbred or selected stocks.
 - Promising new methods for conservation of diploid germplasm, cryopreser-
 8. Funding is difficult to secure for conservation-related research and for the maintenance of live animal collections and germplasm repositories.
 9. It is difficult to predict which stocks will be of value to commercial or academic researchers at some future time. So, despite the uniqueness and potential usefulness of the existing avian genetic stocks, the tenuous nature of the "value" of many stocks has precluded sustained financial support for their preservation from any single agency.
 10. There is currently no organization in the US or Canada to monitor conservation status and availability of avian genetic stocks and set priorities for their conservation on a national or binational level.

Recommendation

An avian genetic resources management system, with strong leadership, but shared responsibility, is proposed as the most efficient and secure way to conserve genetic stocks and address issues of risk and loss. The proposed Avian Genetic Resources System (AVGRS) would be comprehensive and would require the cooperation and collaboration of scientists, funding agencies, and research institutions. The System must be oriented toward research objectives, but it could also support the needs of breeders, breed hobbyists, and breed historians.

Rationale

Historic long-term collaboration between Canadian and US scientists provides a basis for collaboration in the formation and operation of an Avian Genetic Resources System. The US National Plant Germplasm System and its counterpart in Canada serve as excellent models for the proposed Avian Genetic Resources System. A system on this binational level is necessary because no dependable regional or local solution exists.

Components of an Avian Genetic Resources System

The Avian Genetic Resources System is envisioned as a multilocal organization that would serve the avian genetic resources needs for the US and Canada. The AVGRS would feature a central facility as a focal point for many of the activities of the System. The functional components are outlined in Executive Summary Figure 1 and are briefly discussed here.

Avian Genetic Resources Advisory Committee (AVGRAC)

The AVGRS would be advised by this binational committee comprised of representatives of national and state/provincial agencies, stock curators, and researchers. It would consist of 12 to 15 individuals who have worked with avian genetic resource issues, drawn from government, industry, and academic institutions in the US and Canada. Specifically, they should have worked in close association with national, international and private research-oriented organizations, and be familiar with avian genetic stock conservation issues. The members should meet at least once a year and be in regular communication during the year. The Committee would review reports and recommendations for conservation of stocks received from species-oriented committees. It would make recommendations to the management unit of the AVGRS.

Coordination

The various government and research institutions involved in avian research and conservation would use the AVGRS for coordination of information about genetic resources and the AVGRS would in turn be responsible for maintaining and distributing this information. This function would also include strategic planning for conservation of particular stocks, based on advice from advisory groups established for each species. For example, imperiled stocks would be identified to the AVGRS and a plan for their conservation would be developed through coordinated analysis. International relationships would be coordinated through the AVGRS, including conservation of stocks in other countries, import and export of genetic stocks, data sharing, and development of conservation plans for landraces, wild species, and breeding populations.

Conservation

This is the cornerstone activity of the AVGRS and of critical importance. A central facility is needed for conservation and distribution of genetic materials. The central facility would house those living genetic stocks that could not be maintained elsewhere and would serve as a backup site for important stocks that are maintained elsewhere. This would include a secure backup repository for privately owned lines or populations, either as live birds or cryopreserved germplasm at the central or secondary centers on a fee basis. The central facility would also physically maintain the various types of preserved genetic resources and would coordinate those maintained elsewhere. The cryopreservation capabilities of the central facility would be supplemented by a specialized cryopreservation center, presently unused, at the USDA site in Beltsville, Maryland. No site for the central facility is identified at this time.

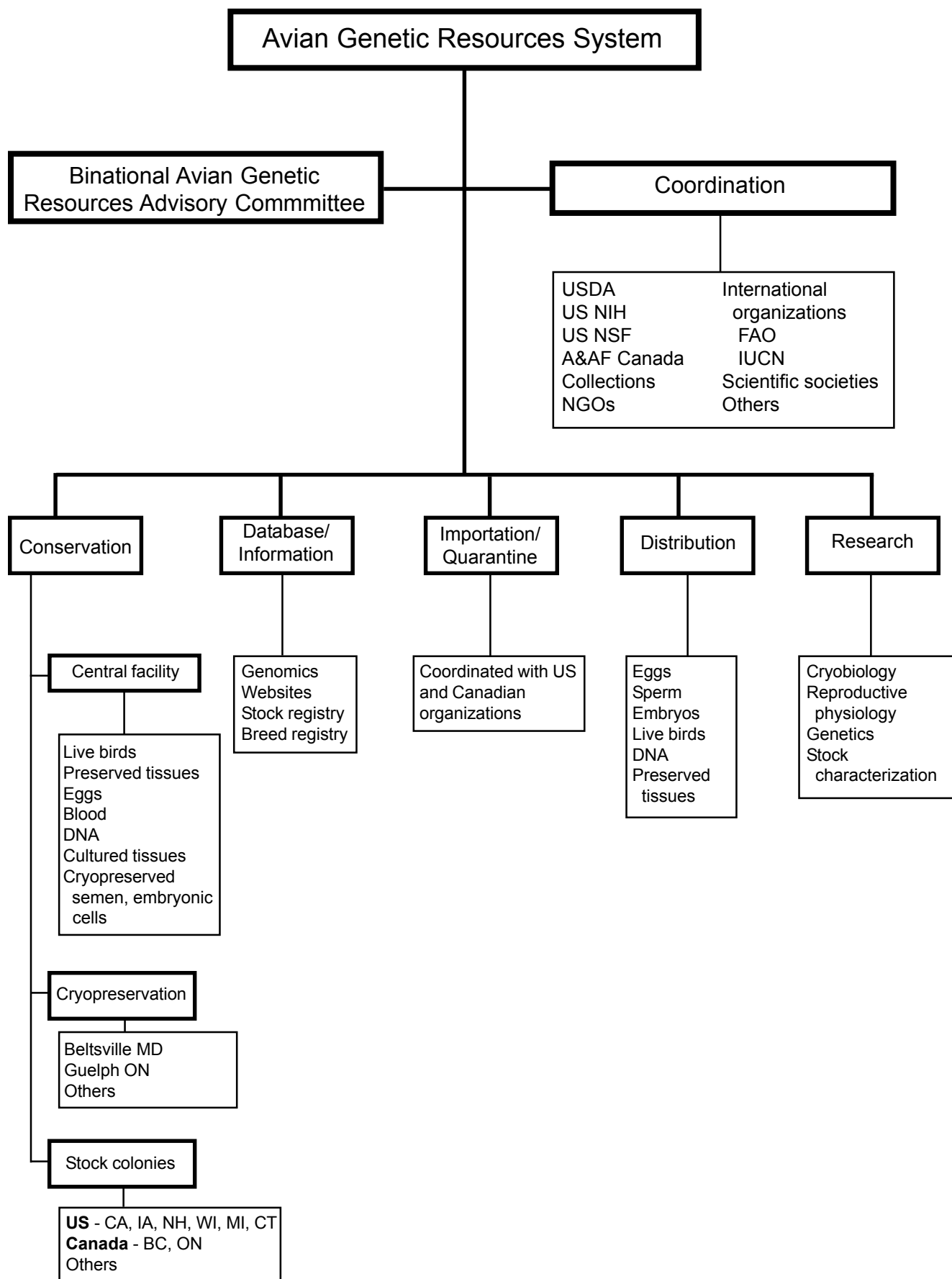
The central facility would support and be linked to secondary facilities located at active research centers which have the capability of maintaining genetic stocks for their own research needs. Several locations in the US and Canada would qualify as secondary sites.

Methods of conservation. Conservation methods employed in the AVGRS would be live-bird maintenance and cryopreservation.

Targets for conservation. Conservation emphasis of the AVGRS should be on live birds, embryonic cells, gametes, DNA, and tissues. The target species for the system will be those of interest in agriculture for food production and for basic biological and biomedical research. Thus, the focus will be on chicken, turkey, and Japanese quail genetic stocks. However, this system could also consider wild turkey, jungle fowl, and game birds, as well as species commonly raised in captivity. The AVGRS will emphasize:

- Genetic stocks having traits and genetic characteristics useful in research, such as inbred lines, single-gene mutations, chromosome aneuploidy, and DNA marker sequences.
- Lines and populations developed by private and public breeders by hybridization and selection for important production-related traits.
- Domesticated mid-level production and fancy breeds held by small producers

Figure ES1. Components of the Avian Genetic Resources System.



and hobbyists in North America and Europe.

- Domesticated, but primitive, landraces existing in Asia, Central and South America, and elsewhere, primarily as scavenger birds.

Archival preserved specimens of birds, organs, skeletons, eggs, feathers, and tissues that have been preserved as museum specimens are also a component of the genetic resources system, since these materials provide for baseline observations and time-course monitoring of factors such as environmental toxicants.

An informal *in situ* system of conservation of landraces and breeds is well established in North America. A monitoring and database system may be the most important need for those genetic resources. This could become an activity of this proposed system.

Databases

Detailed information about all genetic stocks in the US and Canada should be maintained and updated by the AVGRS in a genetic resources information system, similar to the Genetic Resources Information Network (GRIN) developed for the US National Plant Germplasm System and housed with the USDA National Agriculture Library. Additionally, database service would be offered to the various breed conservancies and hobbyists groups for inventory and location of conserved breeds, landraces, and specialty stocks. It would also be logical for the AVGRS database to include DNA sequence data as they are developed.

While GRIN currently focuses on plant information, its goal is to include information on all of the common and endangered breeds of farm animals, including the avian genetic stocks used primarily in research. Collaboration with the AVGRS database would facilitate this goal.

Outreach

Researchers can also be informed of the wide variety of available genetic stocks at the annual meetings of a variety of organizations, including the Poultry Science Association, the Pacific Egg and Poultry Association, the Poultry Breeders Roundtable, the Society for Developmental Biology, the American Association for the Advancement of Science, and the American Medical Association. Presentations at the commercially oriented meetings could be used to showcase the benefits the different companies could derive

from supporting an avian genetic stocks conservation program, while the basic research or disease model aspect of genetic stocks could be emphasized for organizations promoting basic and biomedical research. Thus, the underlying goals of presenting genetic stocks information at such meetings is not only to attract the attention of researchers, but to engage the interest and promote funding from commercial sectors that can benefit directly from research using avian genetic stocks.

Another outreach option for the AVGRS would be an independent website that would promote the available avian genetic stocks to the scientific community by advertising what is available, and indicating those that are slated for elimination. The effectiveness of such a site could be multiplied by linking it with websites: the animal genetic map (Angen), the chicken map (ChickMap), various research organization sites (*e.g.*, Poultry Science Association, Society for Developmental Biology, various commercial poultry sites, and sites for academic institutions).

The AVGRS website information could be further promoted by a series of clearly written review articles in several of the major biological science journals. In each case, a specific area of research would be targeted, such as animal models for human diseases, limb pattern defects, craniofacial defects, integumentary defects, or immunogenetic research.

The outreach activity would also involve international contacts through FAO or various countries with respect to avian genetic resources. For example, there are genetic stocks at risk in other countries that should be considered for rescue in the AVGRS because of their value for research.

Bird care and housing

The housing and care of the live birds at all centers will follow American Association for Laboratory Animal Care standards for a breeding colony. Essentially, the breeding stock should be kept in a facility that approximates that of a well-run commercial poultry breeding farm. The highest degree of automation for feeding, cleaning, watering and climate control is recommended. With most of the chicken genetic stocks, the adult birds will be housed in single-bird cages, and bred by artificial insemination. Other features of the facility may include: floor pens, with or without trap nests, to maintain stocks that do not perform well in cages; and separation of males and the females (in different

rooms or cage rows), so that appropriate, sex-specific breeder diets, lighting, and feeding regimens can be provided for each group.

Importation and quarantine

Movement of animals introduces risks of spreading contagious diseases, obviously of great concern in long-term conservation of live birds. Movement of genetic resources as fertile eggs or semen reduces disease transmission risk and are preferred procedures. Importation of stocks to the central facility will be done through on-site isolation and through national facilities under the direction of USDA Animal and Plant Health Inspection Service. The central facility will have appropriate isolation and sanitation capabilities.

Distribution

A major function of the AVGRS will be to provide genetic materials to users in the research community, breeders, and others. The genetic stocks may be transferred as live birds, semen, or eggs. These will be distributed on a cost-recovery basis. Some users require a continuing supply of genetic stocks and these needs would be supplied by contractual stock reproduction programs. The distribution function would supply well-documented stocks to researchers, thus contributing to the integrity of research projects. This functional component of the AVGRS is analogous to services provided by the Jackson Laboratories for mouse genetic stocks.

Research

The AVGRS should have a research capability within the central facility, especially for developing cryopreservation technologies. Research would also be done on methods for documenting genetic integrity or diagnostics with DNA markers. Other research would be done as needed and appropriate. The research activities would be networked with research laboratories in the US and Canada for collaborative work.

Facilities and organizational aspects of AVGRS

The central facility

Ideally, the primary AVGRS facility would be constructed *de novo* near or part of a major agri-

cultural institution (land-grant university) with a veterinary school that has a good avian medicine program, but reasonably isolated from commercial poultry stocks. The connection with a land-grant institution would give the center close ties to active research laboratories and faculty, who could benefit from such a resource and be drawn upon in support of the center. Access to state-of-the-art poultry disease diagnostics and veterinary care is critical, along with good, off-site quarantine facilities for newly acquired genetic stocks. Locating in a strong poultry producing state would also provide an existing poultry-oriented political and commercial infrastructure that could be mobilized to help support the conservation center.

This facility would include a hatchery, brooding and growing areas, adult bird housing, an isolation area, a cryopreservation laboratory and storage facility, a database center, staff and administrative offices, and a laboratory to support research and analytical services.

Network of secondary genetic stock centers

Secondary stock centers would be designated as part of the AVGRS at land-grant universities and other institutions across the US and Canada that fulfilled two criteria: (1) had adequate facilities and support for the genetic stocks used in its own research programs and (2) had a long-term interest in conserving genetic stocks. Such centers, approved as part of the AVGRS, would receive funding from AVGRS for maintenance of stocks. These secondary centers would maintain live birds and would provide backup for at-risk stocks held in the central facility. As with the central facility, the secondary stock colonies would also have a distribution function on a fee basis. Researchers could also, on a fee basis, arrange to keep research flocks at the central facility or one of the secondary centers, since they may find it difficult or impossible to keep such stocks at their own institutions. Secondary centers could specialize on one or a few classes of genetic stocks.

Management

The central facility and the secondary centers would be administered by the research institutions with which they are located. The central facility would have, at a minimum, a director or manager (research scientist), curator, an administrative assistant, database manager, cryopreservation research scientist, and three or four

laboratory and animal care technicians. The AVGRS would be guided by the advisory committee on all management aspects.

Stock evaluation guidelines

One of the more important activities of the AVGRAC would be the evaluation of stocks for conservation, cryopreservation, or elimination. Guidelines for assessing the value of genetic stocks should be consulted.

Financing the Avian Genetic Resources System

Multiple sources of funding will be necessary to meet all of the needs of the AVGRS. Initial costs are those to construct the central facility and upgrade the secondary stock centers. Annual costs of the central facility would be for its personnel and operations. It would also be necessary to support the annual activities of the AVGRAC. The central facility could also divert funding for specific needs to the secondary centers by means of annual grants.

From the US side, the biological resource programs of the National Science Foundation and the National Institutes of Health would be expected to provide operational funds through direct grants and through grants to investigators who use the avian genetic resources in their research. The USDA's National Genetic Resources System should participate in the AVGRS through the Agricultural Research Service and the Cooperative State Research, Extension, and Education Service. The various State Agricultural Experiment Stations and land-grant and other Universities should also participate. Canadian support and participation should be forthcoming to the extent that the AVGRS provides support to its research and development programs.

The AVGRS will be the major provider of genetic materials to researchers throughout the public and private sectors. For the most part, researchers do not have capacity to maintain live bird colonies and depend upon stock colonies for their research. User-fees are an appropriate means to recoup costs of stock maintenance.

Donation funds can be expected to support the perpetual maintenance of particular genetic stocks. These funds may be provided as annual grants or through income derived from interest on endowment accounts.

Funding should be sought from the US government for construction of the central facility and for personnel support for operations as a part of the US National Animal Genetic Resources System.

Long-term funding would be the most secure from endowment funds. Contributors could be encouraged from the private sector, from large integrated commercial poultry companies to private individuals with interests in preserving poultry stocks or willing to promote the conservation of stocks that can be used to study human diseases.

Construction, personnel, and operational costs have not been established, pending further analysis of potential sites for the central facility and other considerations. For illustrative purposes, rough order-of-magnitude estimates are given in Executive Summary Table 1.

Table ES1. Estimated costs of Avian Genetic Resources System.

Startup costs	
Constructing and equipping central facility	\$15,000,000
Upgrading and renovating secondary centers	2,000,000
Total	\$17,000,000
Annual costs	
Personnel at central facility	\$400,000
Operating costs at central facility	100,000
Grants to secondary centers (8 x \$25,000)	200,000
Advisory Committee	25,000
Total	\$725,000

Avian Genetic Resources Task Force Membership

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Charge to the Task Force

EVALUATE THE CURRENT STATUS of avian genetic stocks maintained in the US and Canada. The assessment should include the following aspects:

- Origin and history of avian genetic stocks.

- How avian genetic stocks have been utilized.

- Value of avian genetic stocks in agriculture, biomedical, and basic biological research.

- Strategies and techniques for long-term conservation.

Produce a report documenting these findings and include recommendations for organizational, fiscal, and administrative steps and personnel necessary to ensure the long-term security of avian genetic stocks for the US and Canada.

Introduction

GENETIC DIVERSITY, IN BOTH WILD and domestic species, is a limited resource worth preserving for future generations (OLDFIELD 1984; ALDERSON 1990; FAO 1992; NRC 1993; BIXBY *et al.* 1994). While many strong advocates promote the conservation of wild species, fewer are aware of the increasing loss of biodiversity in our major food species, particularly among domestic birds. Fortunately, breed conservation organizations have already made some progress in encouraging hobbyists and small-scale farmers in their role as conservators of unique and historically important breeds (BIXBY *et al.* 1994), particularly the less common chicken and turkey breeds (CRAWFORD and CHRISTMAN 1992). These two species are considered more at-risk than most other livestock species (e.g., cow, pig, sheep, goat, or horse) due to recent and extraordinarily rapid expansion of the commercial poultry industry.

In just 50 years, poultry production went from small, individually owned and reproduced farm flocks that formed a small but significant part of the farm income, to the huge commercial meat- (turkey or broiler-chicken) or egg-production ranches that are generally owned or controlled by large corporations (CRAWFORD 1990). In 1997 this industry generated over \$21 billion in poultry products in the US (Box 1 and USDA-NASS 1998).

The intense competition engendered by the rapid growth and often narrow profit margins has served to eliminate the less competitive poultry breeders and to consolidate the high production industrial bloodlines in the hands of a dozen or so poultry breeding organizations. This has created a relatively limited genetic base for the chicken, derived primarily from two breeds (Leghorn and Rhode Island Red) for egg production, and two breeds (Plymouth Rock and Cornish) for meat production (CRAWFORD 1990). A similar situation exists for the commercial turkey. These highly selected industrial stocks considerably out-perform the old production breeds, given the correct feed and management. But the relentless drive to improve the meat- (and egg-) producing abilities of the commercial chicken and turkey stocks has exacted a biological cost: disease susceptibility, leg weakness, muscle defects, and various other inherited conditions that interfere with the ability of the bird to hatch, grow, and reproduce normally (CRAWFORD 1990). Despite these limitations, such stocks have already displaced or diluted some of the hardy, disease-resistant indigenous farm stocks kept in developing countries (MASON and CRAWFORD 1993).

The threat to genetic diversity extends beyond the hobbyist, farm, and commercial poultry

Box 1. Commercial value of the poultry industry

THE CHICKEN AND TURKEY are the two most commercially important poultry species in the US, having steadily increased in popularity with consumers for several decades. In 1987 these two species were the source of over \$12.5 billion in marketable products; in 1997, their total value was \$21.6 billion, derived from almost 8 billion broiler chickens, 300 million turkeys, and 311

million laying hens that produced 77.4 billion eggs that year (USDA-NASS 1998). This placed the 1997 poultry industry below the beef industry in value, but well above the pork or sheep industries, and about on par with the dairy industry.

The Canadian poultry industry is also growing due to both increased consumer demand and a diverse export market. More closely regulated than the US mar-

ket, poultry production in Canada has increased slowly but surely between 1988 and 1996 (the most recent production year available), from 370 to almost 480 million broilers; from 18.6 to 21.6 million turkeys; from 27.5 to 28.2 million eggs. In 1995, the Canadian poultry industry was valued at \$3.7 billion Canadian dollars (approx. \$2.4 billion US) (AA-FC 1996).

stocks. Many of the genetically diverse avian genetic poultry stocks developed, maintained, and studied by academic researchers have disappeared or become at risk in recent years (Boxes 2, 3, and 4, and Chapter 5). As these specialized stocks vanish, unique opportunities for genetic advancement of the different species are lost, and scientific advancement in the agricultural, biomedical, and basic sciences is hampered. Loss of these unusual genetic stocks is not a trivial matter, since the different forms (alleles) of each gene that underlie the observed differences in these stocks may be found only in a single population of birds; if that population is lost, the unique alleles are also lost.

Genetic stocks of chickens, turkeys, and Japanese (Coturnix) quail have played important roles in both basic and applied research, often serving as premier model organisms in the study of fundamental questions in vertebrate biology. In the biomedical field, all three of these species have numerous mutant forms that have provided animal models for the study of certain inherited human disorders (Appendix 2). These include defects such as (glaucoma, macular degeneration) various limb defects, cleft palate, muscular dystrophy, and autoimmune forms of thyroiditis, vitiligo, and scleroderma.

Agriculturally important genetic stocks include those selected for various production-related characteristics. These include egg produc-

Box 2. Dispersal of a genetically significant poultry collection

ONE OF THE LARGEST commercially derived collections of chicken genetic stocks in North America was located at the Center for Farm Animal Research (CFAR) in Ottawa, Ontario. Many informative studies were conducted with historical and modern commercial stocks that dated back to at least 1950. Some of the accomplishments include production of unique control strains from these stocks, improvement of production traits by selection with no evidence of plateaus, study of resistance to diseases including Marek's disease and lymphoid leukosis, comparison of methods of measuring eggshell quality, and the development of semi-

congenic lines for meat-bird-derived endogenous viral genes (GRUNDER *et al.* 1995). The stocks used in these studies included versions of both meat- and egg-selected stocks that had been sequestered from commercial stocks several times since the 1950s, including control strains, multitrait selected strains, and specialty strains selected for one trait such as endogenous viral genes. Unfortunately, the facility in Ottawa lost its government support and was shut down April 1, 1997. Embryonic blastodermal cells from some 30 CFAR stocks not transferred to other institutions were frozen at the University of Guelph (Ontario, Canada).

tion, body shape, feed-use efficiency, leg strength, and disease resistance. Ironically, it is these selected stocks that are particularly vulnerable when funds become limiting, because improvements in production-related traits require many years and a relatively large number of birds each year. Basic research can make use of all these types of stocks, but the various single-gene mutations and genetically uniform inbred and congenic lines have proven to be particularly useful.

Despite recommendations for conservation (CAST 1984; NRC 1990), no formal conservation plan exists in the US or Canada for such research stocks, and many have been lost or now face extinction as curating researchers either retire or lose funding for stock maintenance.

The real and threatened loss of genetic diversity in chicken, turkey, and Japanese quail

Box 3. Collections at risk: University of California-Davis

AN EXAMPLE OF THE PROBLEMS confronting poultry genetic stocks is found at the University of California-Davis (UCD). As with other organizations across the country, UCD has reduced resource allocations for maintenance of genetic stocks. This has become an acute problem for poultry genetic stocks once maintained by the Department of Avian Sciences. The department itself has been subsumed by the Department of Animal Science. The retirements of Ursula Abbott and Hans Abplanalp of the former Avian Sciences Department have put at risk over 50 inbred, mutant, and specialty lines of chickens collected or developed by these two researchers since 1955 (see Appendix 2 for descriptions of these stocks) and

formerly maintained by their research grants and departmental funds. These stocks have been distributed widely to researchers for use in such diverse areas as studies of the immune system (the effects of the major histocompatibility complex haplotypes on disease resistance and the characterization of the physiological parameters of a chicken genetic immune-deficiency syndrome), the architecture of the chicken genome (two of Abplanalp's inbred lines were used to create reference backcross populations and provide baseline DNA for the Chicken Genome Mapping Project), and the effects of inbreeding on different reproductive traits. Some of the mutations are useful as animal models for the study of certain human genetic diseases (ichthy-

sis, muscular dystrophy, scoliosis, and scleroderma). In addition, Abbott has maintained and studied 14 mutations with defined effects on craniofacial and/or limb development and two that affect the integument. Studies with these mutations have provided significant insights into mechanisms controlling vertebrate morphogenesis and pattern formation. These stocks have the potential to contribute significantly to our understanding of a variety of current and future problems within the basic, biomedical, and commercial/agricultural science realms of study, particularly with the use of the rapidly evolving technology that allows researchers to address the molecular genetic basis of such problems.

prompted the formation of the Avian Genetic Resources Task Force (AGRTF). Early in the discussions, Task Force members realized that there were several major, but different, conservation issues. One is the protection of the ancestral wild populations, another is the conservation of unique breeds and landraces of domesticated poultry species, and finally, a more specialized conservation effort is the preservation of unique genetic types developed for use in agricultural, biomedical, and basic biological research, including the various single-gene traits, highly inbred lines, and the populations or lines under improvement for various economic or other defined traits. While the preservation of wild progenitors of domestic species is of critical importance, as is the conservation of nondomesticated avian species world-wide, the Task Force decided that the scope of this report should be restricted to specialized genetic stocks. The Task Force also recognized that rare breeds conservation, as practiced by hobbyists and others, was already in place in North America. The North American poultry stocks currently most at-risk are the unique genetic variants and specialty stocks held by research institutions in the US

and Canada. A comprehensive genetic resources management strategy is herein proposed that can, in the future, provide support and services for all economically important avian species.

This report discusses the history and uses of the different species, presents results of a survey of the genetic resources in the targeted species, gives overviews of major research areas dependent on such genetic stocks, discusses the different conservation methods currently available, and, finally, proposes a plan for a comprehensive Avian Genetic Resource System that would insure long-term maintenance and accessibility of these (and potentially other) endangered avian genetic stocks.

Box 4. Successful conservation of at-risk genetic stocks

THE EXAMPLES OF THREE LINES (Trisomic, from one CSIRO facility (B. Sheldon) to mPNU, and Triploid) serve as models of the traditional mode of curator transfer from originating-investigator/institution to a new curating-researcher/institution. The Trisomic and mPNU lines originated in the Poultry and Avian Sciences Department at Cornell University (S.E. Bloom) and were recently transferred to the University of California-Davis (M.E. Delany). The Trisomic line is also maintained at the University of New Hampshire (R.L. Taylor, Jr.). The CSIRO Triploid line was transferred

from one CSIRO facility (B. Sheldon) to another (R. Pym). Thus, these specialized-cytogenetic lines represent success stories not only because of their significant contributions to basic and applied research, but also in that new homes for the lines were established so that their use and availability for future research are secure, at least for the foreseeable future. Unfortunately, this model of successful transfer of curatorship of genetic lines is the exception today rather than the rule.

Avian genetic diversity: Domesticated species

GENETIC DIVERSITY IS CONSIDERED crucial to the continued survival of a species, be it wild or domestic. Such within-species diversity has been the raw material of agriculturists over millennia. In response to selective breeding and the differential survival of less fit animals, preferred traits have been accentuated and clustered to produce distinct breeds and varieties of the modern domesticated species (NRC 1993). In more recent times, researchers have deliberately isolated various mutations in specialized stocks, permitting the systematic study of such mutations and promoting a better understanding of the normal function of the affected genes.

The totality of wild and domesticated species form the gene pool or genetic resources base necessary for the survival of the species. The genes and genotypes present in this pool represent genetic resources which are accessible and can be exploited by biologists and breeders. In this report, we emphasize “genetic stocks” which have been bred for specific traits and genes in contrast to breeds in which the individual birds have many traits in common and can generally be maintained with randomly breeding populations. Genetic stocks are typically selected for traits of special interest to breeders and geneticists. Many of them are reproductively, physically, or physiologically compromised, and require special care in breeding and management, even for maintenance or conservation purposes.

Target species

While the AGRTF recognizes the need for conservation of undomesticated avian species, this report primarily addresses the need for conservation of specialty stocks of domesticated species, particularly chicken, turkey, and Japanese quail. A limited number of waterfowl (duck and goose) genetic stocks and semi-domestic game-

bird stocks (ring-necked pheasant and bobwhite quail) have been developed and will be noted in this report. Noted below are salient features of the most widely used domesticated species that have the greatest need for conservation of genetic stocks.

Chicken

First domesticated over 6,000 years ago, the chicken (*Gallus gallus* or *G. domesticus*) presents by far the greatest amount of genetic diversity of the domesticated avian species, with over 400 identified genetic variations (SOMES 1988). Many are showcased in the more-than 100 recognized chicken breeds and commercial varieties, which variously integrate most of the naturally occurring mutations affecting size, body type, production characteristics, posture, color, feather structure and location, comb shape, and behavior (see Figures 1 and 2 for wild- and domestic-type chickens). Some of the most extreme variants include: the tiny, short-legged Japanese Bantam; the tall, aggressive Old English Game



Figure 1. Red Jungle Fowl rooster from UCD 001 (Photo courtesy of J. Clark, University of California–Davis).

Fowl; the light-weight Single-comb White Leghorn hen that can lay more than 300 eggs in her first year of production; and the large Rock-Cornish commercial meat chicken, with its phenomenal rate of growth and well-fleshed carcass. These are all thought to share a common ancestor in the Red Jungle Fowl (*G. gallus gallus*) (Figure 1) which is still found wild in parts of India and Southeast Asia (CRAWFORD 1990; FUMIHITO *et al.* 1994), although some poultry specialists believe that several other jungle fowl species (*G. sonnerati*, *G. lafayettei*, and *G. varius*) also contributed to the ancestral gene pool (CRAWFORD 1990).

Turkey

The one commercially important avian species originating in North America, the domestic turkey of commerce, is the product of hybridization between two subspecies of turkey: the domesticated *Meleagris gallopavo gallopavo* from Central America and the wild *M. g. sylvestris* from the eastern United States (CRAWFORD 1990). From these hybrids, birds were selected for size, tameness, carcass yield, and rapid growth, resulting in several distinct breeds and varieties (Figure 3). The modern commercial or exhibition turkeys are large, slow-maturing birds with a much lower reproductive potential than chicken or Japanese quail (at one generation per year for the turkey). Although this species is far less



Figure 2. White Leghorn rooster from UCD 003 (Photo courtesy of J. Clark, University of California–Davis).

Box 5. Parthenogenetic turkeys

PERHAPS THE MOST SPECTACULAR use of turkeys in experimental biology was the study of meiosis, fertilization, and early embryonic development with a strain of parthenogenetic turkeys. In the 1960s and 1970s, M.W. Olsen of the United States Department of Agriculture Agricultural Research Center at Beltsville developed a line of turkeys in which an embryo would form in 30 to 50% of the unfertilized eggs (parthenogenesis). Most of these embryos die, but a small proportion of them (about 0.5%) continue to develop, hatch, and grow into fully functional males (OLSEN 1965). The existence of this line has given rise to the notion that genetic imprinting does not exist in birds, although this conclusion must be tentative in the absence of any formal investigation of imprinting in the unique parthenogenetic stock. However, this parthenogenetic stock exists precariously at only two research stations in the world (the University of Guelph (Ontario, Canada) and the University of Oman).

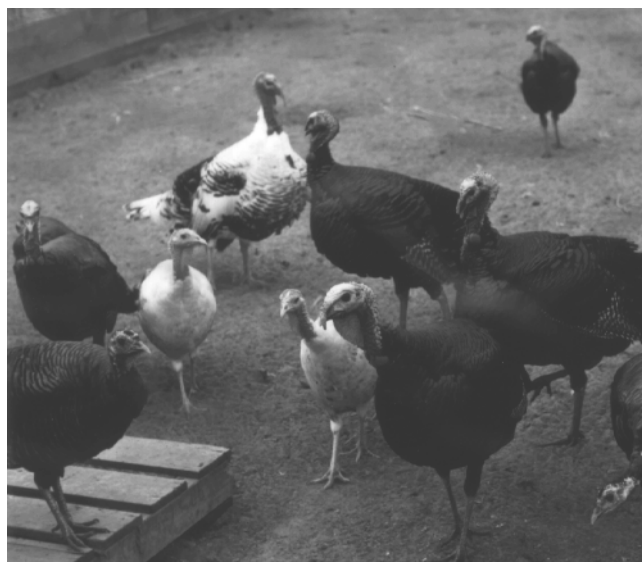


Figure 3. Flock with different turkey breeds (Photo courtesy of F.A. Bradley, University of California–Davis).

popular with researchers or hobbyists than the chicken or the Japanese quail, at least six breeds are still kept for exhibition and a few unique research stocks have been developed (Box 5), including several commercial-type long-term selected and randombred-control lines kept at Ohio State University (see survey results, Appendix 2, Tables 2.1 and 2.2). Perhaps as a consequence of the few researchers studying the turkey, relatively few mutations (49) have been reported in the turkey compared to the chicken and Japanese quail (SOMES 1988).

Japanese quail

Gaining in popularity as an experimental animal in both research and education, the Japanese quail (*Coturnix japonica*) is a small, early maturing, highly efficient egg and meat producer. Until recently, the Japanese quail was classified as a

subspecies of the common European quail (*C. coturnix*). It is now classified as a distinct species because of the nonhybridization of the two in the wild or in captivity (CHENG and KIMURA 1990). According to all available documentation, the domestic Japanese quail strains used in meat and egg production (even in Europe) are descended from *C. japonica*, which is still found in small wild populations in Japan. While gaining popularity as a food animal in the US, its small size has limited its use as a meat- or egg-producing animal to specialty markets. However, the Japanese quail has other qualities that make it ideally suited for research. Usually reaching sexual maturity by six weeks of age, the females often lay an egg a day for several months. The males are aggressive breeders and maintain high fertility even when housed with four or more hens. The early maturity and short incubation interval (16 to 17 days) permit as many as five generations in a single year, in contrast to the slower-maturing chicken (one to two generations per year) or the even slower maturing and less productive turkey (one generation per year). The quail is sometimes called the mouse of the bird world, since it has become extremely popular as a model species for biological research in several fields, including toxicology, cell biology, nutrition, and selective animal breeding. Although most researchers use unselected or randombred birds, over 100 mutations are known in this species, including many affecting feather color and shape (Figures 4 and 5), and several causing embryo-lethal deformities (SOMES 1988; CHENG and KIMURA 1990). At present, most of these mutant strains are only maintained at the University of British Columbia or by hobbyists. Two drawbacks with this species are that the usual

productive life of an individual bird is quite short, frequently less than one year, and, unlike the chicken, close inbreeding is not tolerated. Thus, only one moderately inbred line exists (at the University of British Columbia).

Duck

Almost all of the 15 or so domestic duck breeds recognized today are descended from the wild mallard duck (*Anas platyrhynchos platyrhynchos*), the exception being the Muscovy duck (*Cairina moschata*) (LANCASTER 1990). In addition to the different plumage patterns and colors, a variety of body types and behavioral traits are found among the duck breeds, ranging from the boat-shaped, vocal Call ducks to the cane-shaped Indian Runner ducks. Only 22 mutations have been described in the domestic duck, most involving feather color or pattern (LANCASTER 1990). As such, these traits have been used in defining breed and variety standards, especially among the more ornamental duck breeds, such as the Call, Indian Runner, Crested, Cayuga, and Swedish. While such breeds are usually only kept by hobbyists, a few are important in commercial meat production, particularly White Pekins, Rouens, and, in some areas, Muscovy or Muscovy-domestic duck hybrids.

Goose

Six recognized domestic goose breeds were derived from the western Greylag goose of Europe (*Anser anser anser*). Several other breeds are thought to have descended from the smaller Swan goose of central Asia (*A. cygnoides*). The African breed is believed to be derived from a



Figure 4. Japanese quail *silver* mutation from UBC SI (Photo courtesy of K. Cheng, University of British Columbia).



Figure 5. Japanese quail *porcupine* mutation from UBC PC-WB (Photo courtesy of K. Cheng, University of British Columbia).

hybrid between these two species (HAWES 1990). Strict herbivores, geese have a long history of domestication, but their delayed maturity (two years) and low egg production rate make them less attractive as an experimental animal or as a commercially viable species (Box 6). However, due to the increasingly diverse consumer groups in the US and Canada, formerly noncommercial species are becoming popular on a small scale for specialty markets. One example is the demand from the Asian markets for a smaller, less fatty meat goose. Until now, Europeans and North Americans have traditionally raised Embden geese for market purposes. This large-bodied, fatty bird is not well suited to the method of cooking employed by Asian chefs. Therefore, waterfowl suppliers are now starting to grow the smaller Chinese geese for this market.

Gamebirds

Several species of game birds are commonly bred commercially or by hobbyists, including many subspecies of the Ring-necked pheasant (*Phasianus colchicus*) and the Bobwhite quail (*Colinus virginianus*). Nine color mutations have been identified in the pheasant, along with several affecting skin color and feather structure, and 11 that produce biochemical polymorphisms (SOMES 1990). For most populations, very little selective breeding or inbreeding is deliberately practiced, and the development of gamebird stocks for genetic research is unusual. Exceptions include the now-extinct inbred pheasant lines developed at the University of California-Davis (WOODARD *et al.* 1983) and the Bobwhite

and pheasant blood-type variants currently kept at Northern Illinois University (JARVI *et al.* 1996).

Types of genetic stocks

For the purposes of this report, genetic stocks are classified into four categories that reflect the genetic composition and type and the breeding system used to maintain them.

- Randombred
- Highly inbred
- Long-term selected
- Mutant (including cytogenetic variants and transgenics)

We are primarily concerned in this report with conservation of genetic stocks developed for research purposes, which include all of these categories. Conservation of genetic stocks in the different categories present different challenges for successful conservation, including: high embryonic mortality, low viability, poor reproductive traits, pronounced susceptibility to one or more diseases, large and deleterious genetic load, poor response to specific environmental stressors, poor recovery of cryopreserved semen, and need for a very large gene pool (more than 100 birds per generation).

Randombred lines are maintained as relatively large populations of birds (usually over 100) in which little, if any, selection of breeding stock is done by the curator. Quite simply, the number of progeny from each male or female depends on the reproductive success of that bird at the time the eggs are collected to reproduce the population. Such randombred stocks are generally kept as closed flocks, although new bloodlines may be introduced to the population to improve the vigor of the flock, particularly if inbreeding depression is observed. The birds may be reared and bred in a single large enclosure, with all males having access to all females. This is a common for pheasants, ducks, geese, some chickens, and naturally breeding turkey stocks. Alternatively, the birds may be randomly segregated into smaller floor pens or randomly paired or grouped in cages, as is common with

Box 6. Research with goose breeds in Canada

WHILE GEESE ARE NOT COMMONLY used for experimental purposes, a relatively large experimental population of geese was maintained at the Center for Food Animal Research (CFAR) in Ottawa. Several distinct stocks of Chinese, Embden, and Chinese X Pilgrim hybrid geese were developed in Ottawa to study production traits and DNA fingerprinting patterns (GRUNDER *et al.* 1994). The stocks included standard breed control strains, stocks selected for multiple traits, and the unselected reference strains (SOMES 1988). As with chicken and turkey research in the early part of this century, most studies with the relatively undeveloped pure and crossed goose varieties have been related to agricultural objectives, including methods of rearing broiler geese and improving egg production with controlled lighting and trapnesting. Early studies with Pilgrim geese (the only goose breed that shows strong sexual dimorphism) included a demonstration of increased egg production with selection (MERRITT 1962), and use of light control to increase egg production. More recently, artificial insemination techniques have been improved (GRUNDER and PAWLUCZUK 1991) and geese have been shown to lack endogenous viruses (i.e., viral DNA integrated into the host bird chromosomes) of the avian leukosis type (GRUNDER *et al.* 1993). Unfortunately, with the loss of funding from the Canadian government in April of 1997, these stocks were either eliminated or dispersed.

Japanese quail. A minimum of 25 pairs, usually more than 150 birds, is needed to keep inbreeding at a minimum. These stocks are often kept as a source of “normal” control birds, and also function as a resource stock, from which inbred or selected stocks can be derived or qualitative mutations isolated.

Inbred lines are produced by breeding together close relatives for many generations, resulting in increasingly homozygous and homogeneous progenies. Different types of mating schemes are used, depending on how rapidly the researcher is attempting to approach complete homozygosity. Disregarding parthenogenesis, the most rapid inbreeding is produced by father-daughter, mother-son, or brother-sister (full-sib) matings. These breeding schemes can also be used to expose deleterious recessive traits or to fix preferred or beneficial single-gene traits in a population. Unfortunately, even in the absence of major genetic defects, the fertility and viability of the inbred offspring are almost always lower than the more outbred parent strain, a characteristic called inbreeding depression. If selection and breeding strategies do not compensate for this decline, inbreeding depression can result in the extinction of the line within a few generations. This is a particular concern in lines propagated by full-sib matings which also have large genetic loads (many deleterious alleles). However, once the lethal and sub-vital alleles have been purged from the inbred strain, it can theoretically be bred to essentially complete homozygosity while maintaining reasonable reproductive performance traits (fertility, egg hatchability, egg production rates, viability, etc.). Such genetically uniform stocks can then be used as a standard genetic background in the study of individual genes and gene complexes. Particularly useful inbred lines are those which have been bred for contrasting phenotypes due to allelic differences at single loci. These lines, having the same genetic background for practically all loci except for the alleles of interest, are called congenic lines. They are used to study single-gene effects on productivity, for molecular characterization of genes affecting developmental traits or disease resistance, and many other uses in basic biological and biomedical research (ABPLANALP 1992).

Highly inbred genetic stocks are invaluable in a wide range of research fields, particularly genomics (gene mapping) and immunogenetics (Box 7). A good example of the usefulness of inbred strains in genomics is the mapping of classical mutations. While over 80 classically identified genetic mutations have been assigned to the chicken linkage map, only a few have been located on the molecular map. This is due to the lack of genetic characterization of the exhibition breeds and lines in which most of these mutations are found. Such nonuniform genetic backgrounds make them difficult to use in matings designed to integrate the maps. In contrast, congenic lines, mentioned above, are uniquely useful for such genetic mapping. The integration of genes in exhibition breeds into defined inbred lines would provide the necessary uniformity for molecular mapping of these traits.

Long-term selected stocks are the result of many generations of testing and selective breeding for traits governed by multiple genes (the so-called quantitative or polygenic traits). Many valued heritable characteristics in the poultry breeds belong in this category. These include egg production rate, egg size, feed efficiency, fertility, hatchability, viability, disease resistance, body size and shape, and behavioral characteristics. To change the population mean for one or more of these quantitative traits requires rigorous testing and ranking of the individuals and family groups for the traits-of-interest each generation, followed by selective breeding of the higher-ranked individuals and families to produce the next generation. Many factors can affect the rate of improvement in response to selection including 1) degree of heritability of the trait or traits involved, 2) selection stringency, 3) level of in-

Box 7. Highly inbred stocks in immunogenetics

BASIC INFORMATION ABOUT FACTORS controlling disease resistance in the chicken has been gathered largely from studies with congenic strains of chickens (birds with identical, highly inbred backgrounds but different major histocompatibility complex (MHC) haplotypes; ABPLANALP 1992). A number of these congenic strains have been developed at the University of California-Davis, the USDA Avian Disease and Oncology Laboratory in East Lansing, MI, the University of New Hampshire, and Iowa State University. Researchers have shown how each MHC-haplotype could directly affect the resistance of a bird to a variety of different diseases,

including coccidiosis, Newcastle's disease, and the tumor-inducing viruses that cause Marek's disease and lymphoid leukosis. The congenic MHC strains, most requiring at least ten generations of back-crossing and blood-testing to develop, are key resources required for furthering our understanding of the way the MHC genes function. Studies with these stocks have already given the primary poultry breeders vital information to use in determining the best of several alternative breeding strategies to enhance disease resistance potential of their production stocks.

breeding, and 4) genetic variation in the original source population. Selected stocks usually require several generations to develop, tend to revert towards the original stock values if the selection pressure is lifted (i.e., if random or non-selected pedigree reproduction is used), and usually need to be reproduced in large numbers (several hundred birds) each generation for the best selection differential with minimized in-breeding.

Mutant stocks incorporate one or more of the many single-gene mutations that have a major effect on specific morphological or physiological traits. These include variants (alleles) that affect eggshell color, feather color or shape, skin color, comb shape, metabolic function, major histocompatibility complex (MHC) haplotype identity, and pattern formation in the developing embryo. The wide array of mutations affecting feather color and shape are important for distinguishing between breeds and varieties within breeds. In the poultry industry, eggshell color, skin color, feather color, and feathering rate mutations, and, more recently, MHC types, have all played important roles in the development of commercial strains and varieties. Of particular interest to biomedical researchers are those mutations that cause disease conditions that mimic human genetic disorders, including muscular dystrophy, scoliosis, scleroderma, and a variety of developmental mutations (usually lethal) that affect the development of the face, limbs, integument, and internal organs.

Cytogenetic variants are birds that have chromosomal abnormalities, such as aneuploidy, polyploidy, translocations, and large insertions or deletions. A small number have been established in the chicken, and these have provided useful model systems for the study of meiosis, inheritance, recombination, linkage, transcriptional regulation, and gene dosage effects. Such stocks include: aneuploidy for the chromosome encoding the MHC and nucleolar organizer region (NOR), complete triploidy (three copies, instead of two, of all chromosomes), large deletions (the mPNU line, in which there is segregation of an MHC/NOR chromosome with a deleted NOR), and various stocks carrying translocations between macrochromosomes (Box 8).

Transgenic stocks are formed by inserting foreign

DNA, usually containing a gene of interest, into one of the chromosomes of germline or somatic cells. While some transgenic chickens have been produced in the past few years (SALTER *et al.* 1986; 1987; SALTER and CRITTENDEN 1989), the creation of transgenics is still very much experimental in chickens and other avian species. However, a number of research groups continue to develop and refine transgenic methodologies, and report promising advances in the production of transgenic birds (SALTER *et al.* 1987; LOVE *et al.* 1994; THORAVAL *et al.* 1995; MARUYAMA *et al.* 1998).

Research genetic stocks

Genetic stocks are used in three areas of research: agricultural, biomedical, and basic or fundamental biological research.

Agriculturally important avian genetic stocks primarily include those selected for various production-related characteristics (egg production, body shape, feed-use efficiency, leg strength, disease resistance). Another use for such stocks is to provide a flexible, rapidly responding model system for testing breeding techniques and systems that might also be useful with large livestock species (e.g., pigs, sheep, and cattle). These stocks are particularly vulnerable to funding cuts due to the long development period needed for most selected stocks, and the relatively large numbers that must be produced and monitored annually to produce the selected population.

Biomedical research specifically uses animal models for the study of various human diseases. Avian models, mostly in the chicken, exist for the autoimmune forms of vitiligo, scleroderma, and thyroiditis, as well as for various developmental defects, such as polydactyly, scoliosis, and cleft palate. Genetic stocks are also used in avian health research for studying the nature of

Box 8. Chicken chromosome rearrangement stocks

FROM THE MID-1960s TO THE 1980s, animal genetics laboratories at Ohio State University, the University of Minnesota, and New Mexico State University developed about 40 different chromosome rearrangement strains in the chicken (ZARTMAN 1971; WOOSTER *et al.* 1977; WANG *et al.* 1982). A number of studies by these laboratories made important contributions to our understanding of chromosome behavior in avian species (including recombination, chromosome segregation, identification of pseudoautosomal regions on

the sex chromosomes, and sources of aneuploids). Unfortunately, with the lack of support by various agencies over the last ten years, over thirty of these unique genetic resources were irretrievably lost. The seven still in existence, along with a recently isolated spontaneous translocation, are currently being maintained at the University of Wisconsin. However, with departmental reorganizations and budgetary difficulties, these stocks are also threatened.

disease resistance and effects of specific genes on productivity under disease stresses.

Most of the genetic stocks are of value for studying questions in basic biology that may lead to more applied biomedical or agricultural research, or by simply contributing to the knowledge of how different biological systems function in a wide variety of studies in the life sciences.

Some of the specialized stocks have been derived directly from commercial chicken, turkey, or Japanese quail lines, while others were developed from special breeds, landraces, or wild-types.

Commercial stocks

The commercial poultry stocks have made remarkable genetic progress in the last 50 years (Boxes 9, 10, and 11). At this time, selected stocks used in commercial egg or meat production must fit very specialized production criteria. To develop these criteria, each breeding company has identified particular commercial goals (egg production, weight gain, feed conversion, carcass characteristics, etc.) and seeks to meet them in the shortest possible time (EMSLEY 1993). In this way, the fundamental difference between basic and applied research is highlighted. While a researcher may have a preferred outcome for an experiment, any result can provide useful information to that researcher or others in the research community. For the commercial breeder, the only outcome that is acceptable is one that improves the commercial product for the consumer, and increases final profitability for the producer (HUNTON 1990).

From a commercial production point of view, the loss of unique avian germplasm has a number of negative repercussions. To start with, production objectives and economic standards are constantly changing, particularly for meat production birds. This means that agro-economic industries will continue to need access to genetic diversity to meet future market demands, to adapt to adverse environmental conditions, to fight new diseases, and to meet the demands for different nutritional values. Thus, an effort must be made to identify and conserve all useful genetic resources that could have com-

mercial importance, such as the sex-linked gene controlling the rate of feather growth that has been heavily utilized by modern chicken breeders (Box 9).

In marked contrast to the general perception that commercial poultry stocks all have a relatively small and diminishing genetic base, some researchers have reported the opposite. Specifically, DUNNINGTON *et al.* (1994) used DNA fingerprinting to measure variability among commercial chicken breeding populations and concluded that a considerable reservoir of genetic diversity yet remained. IRAQI *et al.* (1991) reported a great degree of polymorphism for endogenous viral (*ev*) genes in five egg-type populations maintained by an Israeli commercial breeder. AARTS *et al.* (1991) also found variation for *ev* genes among and within six WL and four medium-heavy brown eggshell lines. While none of these methods specifically reflects the variation remaining in genes associated with economically important traits, the recent substantial progress in the development of the genetic map of the chicken (CHENG *et al.* 1995; CHENG 1997) should soon lead to more thorough and realistic assessment of the amount of economic trait variability remaining in commercial poultry populations.

Fancy breeds and mid-level production stocks

For at least 50 years, poultry fanciers have been the main conservators of the majority of the

Box 9. Economics of sex-linked genes and chicken genetics

IN 1908, SPILLMAN REPORTED that the female was the heterogametic sex in chickens (now described as ZW, as compared to mammals where the male is heterogametic, XY). This was based on the finding that the barring gene was inherited as a sex-linked gene, being passed from the dam to her sons. This early finding has played an important role in commercial poultry breeding, as many lines are now sexed at hatch time using the sex-linked rate-of-feathering gene. This gene influences development of the early wing feathers in the chick. If the dam carries the slow feathering mutation, *K*, she passes this on to all her sons, and her *W* chromosome to her daughters. If the sire is pure for the wild type gene, *k+*, all the daughters receive the wild type fast-feathering gene. The male chicks are all slow feathering and the female

chicks are all fast feathering. Such chicks can be easily sexed at hatch time by the relative feather growth by anyone with a minimum of training. Previously chicks were sexed using the vent sexing method, whereby rudimentary copulatory organs were examined to determine sex. This was a costly procedure, and at \$0.03 per chick would cost a hatchery \$3,000 for every 100,000 chicks hatched. Over 600 million egg-type chicks are hatched annually in the US. If only half of these are sexed by feather sexing, the chicken industry saves over \$9 million per year. Broiler breeders are incorporating this gene also, as sex-separate rearing becomes more prevalent. With over 9 billion broilers hatched in the US each year, this also will have an economic advantage to the industry.

poultry breeds and varieties in North America, particularly the old dual-purpose or mid-level production breeds (Box 12). As the Leghorn chicken, Rock-Cornish cross chicken, and broad-breasted Large White turkey became the dominant commercial birds, commercial breeders could see no economic benefit to maintaining other standard breeds and varieties of poultry recognized by the American Poultry Association (APA 1998). Today, without fanciers, it would be very hard to find an Ancona or Silkie chicken or a Royal Palm turkey. The Lamona chicken breed, developed by the USDA, is a notable American example of a once-useful old-fashioned production strain now fallen from favor.

In some cases, access to mid-level stocks can help small-scale producers stay in business. While they cannot compete with the Rock-Cornish meat cross or Leghorn egg-layer in the highly commercial marketplaces, they can become financially successful by raising some of these heirloom birds to supply specialized niche markets (Box 12). There are many other positive

aspects to this practice: small parcels of land can remain agriculturally productive; open space is maintained, family farmers are aided; and moneys go into the local economy.

Biomedical researchers are starting to become aware of the genetic reservoir available in the fancy breeds. They usually seek specific standard breeds or feather patterns that can be used in exploring biological questions (see Chapter 3) or problems related to human medical disorders, e.g., a form of vitiligo in barred chickens (BOWERS *et al.* 1994). The Silkie breed (Figure 9) is particularly useful, with six dominant mutations: crest (*Cr*), rosecomb (*R*), muffs-and-beard (*Mb*), polydactyly (*Po*), ptilopody (*Pt*), fibromelanosis (*Fm*); and one recessive mutation, hookless (*h*). These mutant alleles produce: elongated feathers on the crown of the head (*Cr*) and on the face and chin (*Mb*), a broad, flattened comb that is covered with small, fleshy nodules (*R*), extra toes (*Po*), feathered legs and feet (*Pt*), dark skin, bones, and viscera (*Fm*), and loose, exceptionally fluffy body feathers (*h*). Not only have

Box 10. Development of egg-laying stocks

COMMERCIAL EGG-LAYING chickens (Figure 6) have shown a substantial increase in productivity in the past 60 years. Some of this improvement has been due to developments in the areas of management, nutrition, and disease control, but the effect of genetic improvement is clear (ARTHUR 1986). Between 1940 and 1955, the number of eggs laid per hen in the United States increased from 134 to 192 (USDA-NASS 1998). By 1994, eggs per hen had increased to 254. The change in the

1940s and 1950s was primarily due to the introduction of hybrid stock, utilizing pure breeds which had been under development by numerous small breeders participating in the National Poultry Improvement Plan (NPIP). The more recent increase has been primarily due to selection for increased egg numbers. However, it should be remembered that the work of the small breeders and the formal testing parameters set by NPIP shaped the foundation stocks, paving the way for the phenomenal performance in the modern commercial birds.

Today, only a few large international poultry breeding companies produce most of the world's commercial egg-type chickens. Just 40 years ago, the 1958-59 summary of US random-sample-egg-production tests (ARS 1960) listed 132 breeding firms. In the most recent egg-layer test still conducted in North America, (NORTH CAROLINA COOPERATIVE EXTENSION SERVICE 1996) only five breeding companies were listed. These were actually owned by just three international firms. These three firms breed over

90% of all the egg-type chickens in North America, and probably well over half of the commercial egg-type chickens worldwide.

Crosses among lines of the White Leghorn (WL) breed produce nearly all the commercially marketed white-shell chicken eggs in North America. The WL lines in use today stem from the purebred stocks sold in the 1930s and 1940s. Though there has been intercrossing in many cases to develop new strains, many of the currently used stocks appear to have been selected without intermixing for 30 years or more. Of particular note is the common use of the Mount Hope strain, which is distinguishable by its large egg size and the B-19 and B-21 major histocompatibility complex blood types which it carries (for an explanation of the major histocompatibility complex (MHC) and B-blood types, see the section on Immunogenetics in Chapter 3).

In response to regional consumer preferences, several commercial brown-eggshell chicken lines have also been developed. Typically less efficient than the White Leghorn strains, commercial brown-eggshell chicken lines are usually produced by crossing Rhode Island Red males with high production White Leghorn females. Alternatively, some high egg production strains of Rhode Island Red or Barred Plymouth Rock may be used.

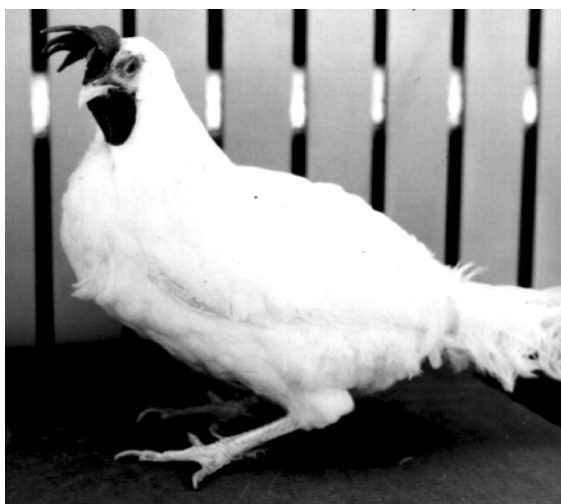


Figure 6. White Leghorn hen (Photo courtesy of U.K. Abbott, University of California-Davis).

the hobby breeders helped in supplying such research birds for one-time projects, but some of them have participated in long-term breeding programs for researchers.

Although the majority of exhibition and mid-level production poultry breeds have continued to exist under the rather informal stewardship of

the hobby breeders and the different breed organizations, a number of problems are associated with their conservation: 1) most of the amateur conservators often only keep their stocks for a short period of time (typically just five years); 2) small-scale hobby breeders who get breeding stock from a central clearing house of poultry

Box 11. Development of meat-producing stocks

IN 1950, A COMMERCIAL BROILER took 84 days to grow to 1800 grams; by 1970, this was cut to 59 days, and by 1988, it was down to 43 days (from HUNTON 1990). As with the egg-type chickens, a large proportion of the improved performance of meat birds can be attributed to developments in the areas of management, nutrition, and disease control. But choice of foundation breeding stock and early use of breed crosses were also important in the development of the broiler industry.

More so than the egg market, the broiler market is strongly consumer-driven (POLLOCK 1999). Early consumer input (chicken of tomorrow competitions between 1946 and 1948) gave the broiler-breeders and growers a good picture of consumer preferences: compact, well-fleshed carcasses at affordable prices. In other words, the scrawny, angular cockerels (Figure 7) available in large numbers from egg-selected Single-comb White Leghorn lines did not even approach the con-

sumer ideal. The broiler-breeders were fortunate to have available the Cornish breed (derived from fighting stock imported from India), which had many of the desired carcass characteristics. The breeders also found that the production characteristics (body type, rate of gain, feed conversion) improved rapidly in response to selection. Unfortunately, improvement in these areas had a strong negative effect on the already poor reproduction characteristics of the Cornish lines (low egg production, low fertility, poor hatchability, reduced chick viability), and seriously impaired disease resistance (see section on immunogenetics, Chapter 3). The early breeders found that crossing the Cornish roosters with hens from improved dual purpose breeds solved many of these problems. These "female" line breeds, including the Plymouth Rock and New Hampshire, have better body type than Single-comb White Leghorns, yet lay eggs at a relatively high rate compared to the Cornish "male" lines. Today, the commercial sire is often a cross between two predominantly Cornish strains, and the commercial dam is a cross between two strains descended from one or more

of the dual-purpose breeds. The outbred or crossbred parents have better reproductive traits and general vigor than parents from the pure-lines, and their offspring, a three- or four-way cross, exhibit even more hybrid vigor. Unfortunately, despite careful evaluation of the breeding stock, some serious structural and physiological problems have surfaced that appear to be the result of the intense selection for desirable production characteristics. These include: leg weakness, cardiopulmonary insufficiency, breast blisters, increased fat deposition, and muscle anomalies.

While the turkey industry is considerably smaller than the broiler chicken industry, many of the same breeding methods have been used, and many of the same problems have been encountered (HUNTON 1990). With a smaller genetic base, and a much larger bird to start with (Figure 8), the structural and physiological problems found in chickens are often magnified in turkeys. Considering the small number of primary breeders (three) and the scarcity of exhibition or research turkey breeding stock, it is imperative to safeguard the remaining genetic diversity of this domestic species.



Figure 7. Traditional broiler-type chicken carcass of the 1940s (Photo courtesy of F.A. Bradley, University of California–Davis).



Figure 8. Commercial Large White turkey tom (Photo courtesy of R.A. Ernst, University of California–Davis).

stocks may never know their egg source or the degree of relationship of their foundation stock; 3) breeding populations are often very small, particularly for the rarer breeds, and pedigree information is frequently limited or not available; 4) some hobbyists deliberately inter-cross different breeds or varieties in attempts to improve or modify exhibition traits; 5) selection for production characteristics (e.g., fertility, viability, egg production, or disease resistance) may be largely ignored in these small-scale breeding programs, although *de facto* natural selection will tend to eliminate the infertile, disease-susceptible, or least-viable individuals; and 6) backyard breeders tend to have problems in controlling diseases and may have serious endemic diseases. If there were a formal conservation program for avian genetic resources, it would be logical for it to provide technical services to these hobbyists who are a very important component of avian genetic resources conservation.



Figure 9. Silkie rooster from UCD Silkie (Photo courtesy of J. Clark, University of California–Davis).

Box 12. Small renaissance of old-style chicken breeds

WITH THE INCREASING CULTURAL diversity of our population, the white-feathered, highly selected meat- or egg-producing bird no longer meets the needs of all consumers. In response to a great demand by ethnic markets and the many upscale restaurants searching for the “chicken of yesterday”, more and more small producers are starting to raise “old fashioned” mid-level production or dual purpose breeds (those that are reasonably efficient at producing both meat and eggs). These produc-

ers are getting their stocks from the few people who still maintain populations of true Rhode Island Reds, Speckled Sussex, New Hampshires, and so on. Those supplying the specialty egg markets are also looking for different breeds to produce a colored egg that will be distinctive (brown, tan, green, or blue), such as Orpington, Rhode Island Red, and Ameraucana. Unfortunately, most of these so-called mid-level production breeds have all but disappeared from American farms.

Avian genetic resources for biological research

ALTHOUGH MOST WIDELY KNOWN for their utility in the production of food (meat and eggs) and other commercial products (feathers, vaccines, fertilizer, etc.), chicken, Japanese quail and other domesticated avian species have long been a preferred experimental organism in applied and basic life sciences. Some features contributing to their popularity are:

- Availability (poultry, especially, are easily raised and readily obtained, with inexpensive common strains providing cost-effective research animals).
- High rate of embryo production compared to mammals.
- Accessibility of the avian embryo (the externally incubated avian embryo is available for experimental manipulation during most stages of embryonic development).
- Excellence as a higher vertebrate animal model.
- Ease of surgical manipulations (surgical procedures on birds are much less likely to result in infections due, perhaps in part, to their high body temperatures).
- Extensive history of use (more than a century of research provides avian scientists with needed information and genetic stocks to ask specific experimental questions).
- Animal welfare considerations are generally less complex for domestic birds than they are for other vertebrates.

Research with a variety of genetic stocks has produced many important discoveries in the areas of genetics, virology, immunology, developmental biology, and animal agriculture. (see CRAWFORD 1990, for a review). These include the pioneering studies on Mendelian inheritance and sex determination of BATESON (1902), and BATESON and PUNNETT (1906, 1908). Not surprisingly, given its history of use in genetic studies, the first linkage map reported for any domestic animal species was that for the chicken published by HUTT (1936). The turkey has provided one of the few known vertebrate models for parthenogenesis (embryo formation in an unfertilized egg) (Box 5), and the wide range of metabolic and developmental mutations in chicken (Boxes 8, 13) and quail have allowed researchers to start investigating the molecular bases of these defects, and, eventually, to determine the normal function of the affected gene. In virology, chicken hosts were used by ROUS (1911) to identify the first tumor-causing virus, Rous sarcoma virus. Later, the genetics of host resistance to RNA viruses was elucidated (CRITTENDEN *et al.* 1963), and the Marek's-like herpes virus found in turkeys was used in the first successful vaccination program against a tumor-causing virus in the chicken (OKAZAKI *et al.* 1970). More recently, BUMSTEAD and PALYGA (1992) described one of the first molecular genetic maps of a domestic animal using DNA markers (RFLPs), and the chicken genome map continues to be among the best developed of those for agricultural animals (LEVIN *et al.* 1994; CHENG *et al.* 1995; CROOIJMANS *et al.* 1996; CHENG 1997). It is by far the best-developed map for any avian species or any species with ZW sex chromosome determination. In particular, the usefulness of the chicken genome map in cross-species comparisons was highlighted at the First International Workshop on Comparative Genome Organization (ANDERSSON

et al. 1996), although workshop participants also expressed concern that the continuing loss of poultry genetic stocks will impair some important comparisons with the human and other genome maps. The chicken gene library was the first one described for an agricultural animal species and, along with the human gene library, was the first constructed for any vertebrate (DODGSON *et al.* 1979). Critical early observations of gene structure, organization, and expression were made using chicken ovalbumin (WOO *et al.* 1978), globin (DODGSON *et al.* 1979), histone (ENGEL and DODGSON 1981), insulin (PERLER *et al.* 1980), and lysozyme (STEINER *et al.* 1987) genes, among others.

Avian reproductive biology

Birds such as the chicken are uniquely adapted for the production of large numbers of offspring, in the form of fertile eggs, over relatively long periods of time. In the wild, birds will usually lay a series of eggs on sequential days until they have as many eggs as they can comfortably incubate (a "clutch"). At that time, they will stop laying eggs and start to incubate or brood the eggs. However, during their long domestication, chickens have been selected for large clutch size and nonbroody behavior, so that hens today will continue to lay at a high rate for at least a year,

given proper conditions. In fact, chickens from some commercial stocks that have been selected for egg production may average over 280 eggs in a one-year period, with fertility of greater than 95%. While genetic strains used in research are usually less productive, most produce at least 100 eggs per hen per year, providing researchers with an abundant, dependable source of individually packaged embryos of known genotype.

The functional reproductive system of most female birds, including the chicken, is composed of the left ovary and the oviduct. The ovary contains ova in varying stages of development, with the largest being released at regular intervals. The oviduct is a tubular organ, consisting of several parts: the funnel-shaped infundibulum, where each ovum enters the oviduct and fertilization occurs; the wide, thick-walled, magnum, where the albumen is produced and deposited around each of the ova; the narrower, thinner-walled isthmus, where the shell membranes are made; the large, pouch-like shell gland, where the egg is "plumped" with fluids and electrolytes, before the calcium carbonate shell is deposited on the shell membranes; and, finally, the vagina, with its specialized sperm storage sites at the utero-vaginal junction, where spermatozoa can remain viable for more than a week (VAN KREY 1990; BAKST 1993). Thus, the oviduct is where both the fertilization and the packaging of ovum

Box 13. Inherited muscular dystrophy of the chicken

GENETIC MUSCULAR DYSTROPHY (GMD) of the chicken was first formally described in 1956 in selected New Hampshire strains at the University of California-Davis. Since then, literally hundreds of reports have been published on the abnormality. The disorder is caused by a single gene; but little is known of the molecular nature of the defect (PESSAH and SCHIEDT 1990; WILSON 1990; IANNACONE *et al.* 1995).

Symptoms of the disorder typically appear during late embryogenesis and neonatal muscle maturation. Muscles with fast twitch, alpha-white muscle fibers such as the superior pectoralis and biceps are most affected. These "white" muscle fibers exhibit irregular-shaped rounded fibers, both larger and smaller than normal. Eventually the muscles atrophy and are replaced by fat and connective tissue. The early appearance of GMD in fast twitch but not in tonic fibers suggests it is expressed in a particular myogenic lineage.

Studies have shown that the problem with dystrophic muscle is not a faulty set of neural instructions. Limb

bud transplants demonstrated that dystrophic muscles grafted to a normal host and its nerve were phenotypically dystrophic, and normal muscles transplanted to a dystrophic host were normal, indicating the problem was in the muscle and not directly its innervation. Thus, if neural influences are involved in GMD, it is likely it is the dystrophic muscle that is unable to respond properly to signals from its phenotypically normal nerve.

One of the major uses for the dystrophic chicken has been to test therapies, including an elevated oxygen levels (ASHMORE and SOMES 1966), the drugs D-penicillamine (which affects collagen formation; PARK *et al.* 1979), cyproheptadine, methysergide, and diphenylhydantoin. A Muscular Dystrophy Association-supported drug screening program achieved promising results with steroids such as corticosterone-21-acetate (C21A). The most effective treatments severely reduced the growth rate of the chicks, as if the synthesis of defective proteins play a role in expression of the abnormality.

The discovery of and sequencing of dystrophin, the gene associated with Duchenne muscular dystrophy in the human and the *mdx* dystrophy in the mouse, added a new dimension to the study of muscle abnormalities. The dystrophin protein itself is absent in humans with Duchenne dystrophy and in the *mdx* mouse. This led the Muscular Dystrophy Association to focus its efforts on the human and mouse forms of the disease. But it is still not known whether or not the dystrophin gene product is defective in GMD of the chicken. It is known that dystrophin in normal chickens is closely homologous to the mammalian gene, and that a dystrophin protein is present in GMD chickens. It is not known if this protein is the same as the one in normal chickens. Future research focusing on the role of the dystrophin gene, establishing expression of the abnormality in cell culture, and studying the factors that regulate maturation of normal muscle are worthy of study.

occurs, producing the neat, aseptic association of nutrients and germplasm that will ultimately form the chick. Embryonic development progresses as the egg moves down the oviduct, so that when the egg is laid 24 to 27 hours after fertilization, the embryo is a radial dome of 40,000 to 60,000 cells that is already forming distinct layers (epiblast and hypoblast). At this stage of development, the embryo is comprised of cells that are morphologically undifferentiated (EYAL-GILADI and KOCHAV 1976; WATT *et al.* 1993) and pluripotent, i.e., able to differentiate into a number of different cell types (PETITTE *et al.* 1990).

Numerous studies on avian reproduction have been conducted since the turn of the century, particularly on the control of fertility and egg production (HUTT 1949; LERNER 1950, 1958). Consequently, much is known about genetic and environmental factors that influence or govern reproduction in the male and female chicken, turkey, and Japanese quail. Some of the genetic variants affecting reproductive traits that were studied include: restricted ovulator, multiple ovulator, riboflavin transport deficient, and male infertility associated with the rose-type comb.

Embryology

There is a long and rich history of use of the avian embryo as a model system for study of vertebrate development (ROMANOFF 1960). Beginning with Aristotle (384–322 BC) whose *Historia Animalium* includes the first known fully preserved description of a chicken embryo, researchers and natural science historians have found bird embryos to be a readily available, dependable source of embryos that could be manipulated and observed during the course of development independent of and without harming the female parent (a major issue when studying mammals). Its easy access during organogenesis, relatively inexpensive cost, and bilaterality providing opportunities for contralateral controls for microsurgery and other perturbations have made the avian embryo one of the best characterized vertebrate models, both in classical and molecular terms (MACCABE and NOVEROSKE 1997; MORGAN 1997). Another factor that has made the avian embryo the model of choice for studies of vertebrate development is the respectable catalog of existing mutations (listed in ABBOTT 1967; SOMES 1988; and CRAWFORD 1990). Avian embryos have also proven uniquely adapted to interspecific tissue grafting because of distinct

differences between the appearance of chicken and Japanese quail cell nuclei. Selected early embryonic cells from quail have been grafted into chicken blastodiscs, which, after careful incubation, reveal exactly how the donor quail cells are distributed in the later embryos (DIETERLEN-LIEVRE and LEDOUARIN 1993; DIETERLEN-LIEVRE 1997). Such studies have permitted the development of a detailed fate map of the early blastodisc. Additionally, avian embryos have been used extensively in teratological studies, in which their response to chemical, nutritional, hormonal, and environmental challenges were evaluated to provide guidelines for identifying causes of nongenetic developmental defects (LANDAUER 1967, 1973), and, eventually, for identifying the biochemical mechanisms disturbed by such exposures. Avian embryos have also been of value in studies of cell proliferation related to cancer-like cell growth or programmed cell death inherent in certain stages of embryonic development (SANDERS and WRIDE 1997).

Models for human genetic diseases

Animal genetic defects have long been used as model systems in the study of human diseases. A few of the avian genetic disorders that have proven especially useful include the autoimmune forms of avian vitiligo (AUSTIN *et al.* 1992; AUSTIN and BOISSY 1995; ERF *et al.* 1995), scleroderma (GERSHWIN *et al.* 1981; ABPLANALP *et al.* 1990; VAN DE WATER *et al.* 1994; SGONC *et al.* 1995), and thyroiditis (reviewed by ROSE 1994), the polygenic avian scoliosis (RUCKER *et al.* 1986; LIEN *et al.* 1990; MOCHIDA *et al.* 1993), the autosomal recessive form of muscular dystrophy (PESSAH and SCHIEDT 1990; WILSON 1990; IANNACCONE *et al.* 1995) (Box 13), and a polygenic muscle defect in the broiler chicken (Box 14). Other known avian mutations mimicking human disorders include: autosomal and sex-linked dwarfism, lamellar ichthyosis, poly- and hypodactyly, gouty uremia, genetic obesity, several types of micromelia, glaucoma, a non-autoimmune form of vitiligo (BOWERS *et al.* 1994), and a variety of neurological disorders (SOMES 1988; CRAWFORD 1990).

Japanese quail disease models have also attracted the attention of biomedical researchers. For example, at the University of British Columbia, quail serve as a model in the study of atherosclerosis, and in age-related macular

degeneration (AMD). This condition is the most common cause of blindness in humans over the age of 65. Except for primates, the quail is currently the only suitable nonhuman species in which to study this disease.

Despite the proven utility of existing strains of chickens, turkeys, and Japanese quail in biomedical research, fewer and fewer researchers are making use of these unique genetic stocks (WILSON 1990). Contributing to this decline are the increasing costs and the more technical (and costly) nature of many studies that have biased researchers towards smaller vertebrate laboratory species (rats and mice). Whether or not it is the best possible choice, the default condition for medical research continues to be the rodent, ultimately limiting the study of disorders and diseases to those suitable to such animal models. Enhanced use of exceptional avian models depends on the continued accessibility of existing stocks and the development and maintenance of new genetic stocks.

Immunogenetics

Natural genetic variation in the major histocompatibility complex (MHC) has been studied extensively in the chicken, particularly as it relates to disease resistance (GUILLEMOT *et al.* 1989; BACON and DIETERT 1991; HELLER *et al.* 1991; SUNG *et al.* 1993; KEAN *et al.* 1994; SCHAT *et al.* 1994; BACON and WITTER 1995; HEMENDINGER *et al.* 1995). The different MHC haplotypes have been systematically integrated into highly inbred stocks to control or eliminate complications in immune response introduced by subtle differences in the genetic background (ABPLANALP 1992; Box 7). These studies led to a better un-

derstanding of the complexities of the vertebrate immune system. They have also provided the framework for the development of disease-resistant poultry stocks (LAMONT 1994) and for understanding the differential effectiveness of certain vaccines in chicken strains with different MHC haplotypes (BACON and WITTER 1992).

In studies which focused on cell-mediated immunity and macrophage function, researchers showed that genetic differences between selected strains can also affect the ability of a given bird to promote or suppress Rous sarcoma tumor formation (QURESHI and TAYLOR 1993).

The relationship between MHC haplotype and immunocompetence is currently attracting the attention of the commercial sector. This is due to a strong negative correlation between characteristics such as improved feed conversion and more rapid growth rates with a weaker antibody response to specific antigen challenges and a decreased effectiveness of cell-mediated immunity, both of which are related to MHC function (GAVORA 1990; HELLER *et al.* 1991). This trend in immune responsiveness of commercial egg-laying Leghorn strains (small, slower-growing birds) is compared with that of commercial broiler strains. In addition, a distinct loss of immunocompetence was shown in a study that compared two broiler-type lines, one descended from commercial stocks developed in Canada in 1957, and the second from modern commercial stocks (QURESHI and HAVENSTEIN 1994). Thus, commercial broiler-chicken breeders are now showing interest in improving immune system function to offset the immune depression that has been bred into their stocks with long-term, intensive selection for meat production traits.

Box 14. Deep pectoral myopathy: One price of intensive selection

DEEP PECTORAL MYOPATHY (DPM) is characterized by death of the mid-region of the supracoracoideus muscle of the large broilers and marketable turkeys. It is also known as "Green Muscle Disease" and "Oregon Muscle Disease".

A series of investigations by Siller, Harper, and their colleagues (WIGHT and SILLER 1980; WIGHT *et al.* 1981; HARPER *et al.* 1983; SILLER 1985; WILSON *et al.* 1990) demonstrated that the myopathy was due to an ischemia caused by swelling of the muscle during exercise. An enlarging muscle, an inelastic muscle fascia, and a rigid sternum create a situation in which circulation is cut off to the mid-region of the muscle. Ultrastructural changes were detect-

able in as little as 15 minutes when broilers were induced to flap their wings for short periods of time.

Polygenic physiological problems are not simply resolved. Food preferences of the public and the economic forces of the poultry industry favor selection for large breasts maintaining a physiological situation in which deep pectoral muscles predisposed to ischemia may continue to be a problem. Indeed, SILLER (1985) said that the disorder was a "penalty of successful selection"; "...wild turkeys and less intensely selected old commercial strains are apparently not susceptible to DPM, it is obvious that this disease is man-made". One solution would be to breed broilers and turkeys

with better circulation and a different breast muscle configuration still acceptable to the public which would probably require outcrossing to the unaffected, unimproved genetic stocks.

DPM has its human counterpart. It is one of a class of pathological situations known as "Compartment Syndromes". One of these is anterior tibialis syndrome or "March Gangrene" in which hard exercise by untrained or susceptible individuals (such as military recruits on their first forty-mile hike of basic training) results in permanent muscle damage similar to that found in turkeys and large broilers.

As more is learned about the genetic mechanisms controlling disease resistance and susceptibility, researchers will be able to narrow their focus to specific genes. A few of these genes are now known, but the majority remain to be identified. As with plant genetic stocks, some resistance genes may well be found in stocks not now commercially useful, e.g., wild or feral populations and breeds such as Ancona chicken (Box 15) or other relatively unimproved populations (GAVORA 1990).

Cytogenetics

The chicken has proven to be a good vertebrate cytogenetic model, despite the complexity of its karyotype (the majority of the 78 chromosomes are exceptionally small microchromosomes; see Figure 10). In particular, unique cytogenetic conditions in the chicken have encouraged biologists to use the chicken as a means to study altered chromosome constitutions (e.g., gene dosage effects) on the biology of this and other higher vertebrates. The chicken is exceptional among the higher vertebrate animal group in that several viable genetic strains exist which showcase the effects of chromosomal aneuploidy, polyploidy, or large deletions, several of these occurring in microchromosomes.

The linkage and chromosomal location of the major histocompatibility complex (MHC) with the single nucleolar organizer region (i.e., the rDNA locus) were determined in studies of birds from the Trisomic Line (BLOOM and BACON 1985),

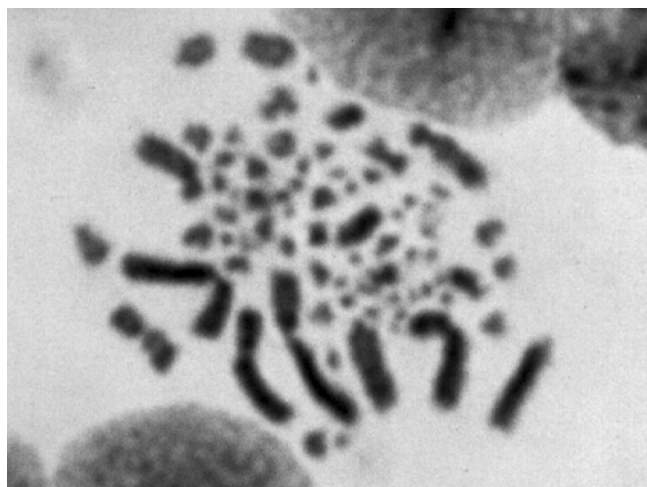


Figure 10. Chromosomes from a Red Jungle Fowl hen from UCD 001 (Photo courtesy of M.E. Delany, University of California–Davis).

Box 15. Ancona chickens give a new angle on disease resistance

AN UNUSUAL GENE CONFERRING disease resistance was recently discovered in the rare Ancona breed of chicken. A unique copy of a gene outside of the known major histocompatibility complex (MHC) was identified that imparts resistance to the commercially troublesome Marek's disease virus. This gene, *Rfp-Y*, now also identified in other chicken breeds, is on the same chromosome as the MHC, and is still being characterized (BRILES *et al.* 1993; WAKENELL *et al.* 1996; MILLER *et al.* 1994, 1996).

which includes individuals with two (normal or disomic), three (trisomic), or four (tetrasomic) copies of chromosome 16 (a microchromosome). The biological consequences of aneuploidy and gene dosage effects on growth, development and immunity have been the subject of many studies (DELANY *et al.* 1988; QURESHI *et al.* 1989; HEMENDINGER *et al.* 1992; LEPAGE *et al.* 1996) and continues to be important in current research. The Trisomic Line has been particularly useful in studying chromosome dosage effects on regulation of MHC expression, with tetrasomics and trisomics expressing distinctly higher levels of MHC-products on their cell surfaces than disomics (MUSCARELLA *et al.* 1987; DELANY *et al.* 1988; HEMENDINGER *et al.* 1992).

The mPNU line segregates for a large deletion in the nucleolar organizer region of chromosome 16, giving it a reduced number of rRNA genes (DELANY *et al.* 1995). This line was used to establish the developmental threshold (i.e., lethal limit) for rRNA gene copy number for the first time in a higher vertebrate (DELANY *et al.* 1994, 1995). The Trisomic and mPNU lines were instrumental in establishing the location of a new locus, *Rfp-Y* (Box 15) on this microchromosome (MILLER *et al.* 1994, 1996).

The CSIRO Triploid line, maintained in Australia, is an important higher vertebrate polyploid model. Studies of the line have contributed information regarding errors of meiosis, genetic basis for inheritance of meiotic errors, sex determination, gonadal differentiation, sex reversal, and polyploidy effects on growth and development (LIN *et al.* 1986; THORNE *et al.* 1987, 1988, 1991, 1997; SOLARI *et al.* 1991; THORNE and SHELDON 1991).

Genomics

Genomics is a rapidly evolving field of science that emphasizes the study of complete genomes and chromosomes (Box 16). Genomic studies have also changed in focus, from the general organization of the genome to the identification and localization of functional genes. Genomic research is well advanced with the chicken.

Genome map development involves coordinated approaches (BURT *et al.* 1995): 1) expanding and improving the genetic linkage maps of chickens and other poultry, 2) using markers, such as microsatellites, that have high utility, improving the cytogenetic maps available for birds, and 3) making new efforts towards integrated physical (recombinant DNA clone-based) maps. Candidate gene research involves a variety of genes of interest, including those encoding the major histocompatibility complex genes, other immune-response genes, ribosomal RNA genes, cytokine and other hormone-encoding genes, and genes related to muscle and morphology. Finally, several efforts are underway to map and identify anonymous genes associated with disease resistance, growth and reproduction, and general viability of poultry.

The interdependence of genome research and genetic resource conservation can hardly be overstated. Inherent to all genetic experimentation and breeding is the fundamental requirement for allelic diversity. It is preferable that diverse alleles be preserved in well-characterized, inbred (homozygous) lines. As a concrete example, it is widely agreed that one of the reasons that the laboratory mouse has become the most important model system for biomedicine has been the availability of diverse inbred lines (pioneered most notably by the Jackson Laboratory). COPELAND and JENKINS (1991) used such inbred mouse strains in their intraspecific backcross reference family approach to gene mapping. A similar strategy was followed in developing the East Lansing chicken mapping reference population, derived from the segregating progeny of a cross between two inbred lines: Red Jungle Fowl (UCD 001) and White Leghorn (UCD 003), genetic stocks developed by H. Abplanalp at the University of California, Davis (CRITTENDEN *et al.* 1993). The two lines are highly divergent, displaying a high degree of interline polymorphism. The diverse and inbred character of these lines has been a major contributor to their subsequent utility. In contrast, BUMSTEAD and PALYGA (1992) used animals from two divergent Leghorn lines as the parents for their reference backcross panel. Cheng and Bacon (Avian Disease and Oncology Laboratory,

personal communication) have used inbred lines (RPRL 6I2 and 7I3) for mapping of non-MHC genes which are responsible for resistance/susceptibility to Marek's disease virus. More generally, BACON's (1987) congenic MHC lines have been critical for a variety of genetic analyses of the avian immune system. Again, the speed with which this work was accomplished was enhanced and it could only have been accomplished using the available well-characterized genetic lines.

One long-term objective of avian genomics is to develop an understanding of genes responsible for economically important traits in poultry. These are variously known as quantitative trait loci (QTLs) (VERRINDER GIBBINS 1993) or economic trait loci (ETLs). The ETLs are analogous to, and sometimes models of, complex genetic traits of humans and other species. In some cases, the protein product of these genes is already known. In other cases, researchers seek to identify and further characterize previously unknown ETLs (anonymous genes). Among the genes under study are those encoding disease resistance traits. These include major histocompatibility genes, cytokine genes, and anonymous genes encoding Marek's disease virus resistance. Other genes of interest include those which regulate growth, reproduction and morphology (e.g., endocrine genes, collagen and other bone and connective tissue genes, anonymous ETLs), and genes which relate to general viability and cellular function (e.g., ribosomal RNA genes,

Box 16. Government involvement in genome research

THE US GOVERNMENT IS HEAVILY invested in genome research. In response to the call for a USDA National Genetic Resources Program in the 1990 Farm Bill, the USDA created the National Animal Genome Research Program (NAGRP), administered by Cooperative State Research, Education, and Extension Service (CSREES) with Richard Frahm as Director. The NAGRP was developed as an equal companion to the National Animal Germplasm Program (NAGP), administered by Agricultural Research Service (ARS) with Roger Gerrits as director. The essential interrelationship of genome research and germplasm resources is emphasized by the common origin of and close ties between the NABRP and the NAGP. Support for agricultural animal genomics comes primarily from the USDA as direct support to ARS laboratories, such as the

Avian Disease and Oncology Laboratory (ADOL) in East Lansing, the National Research Initiative Competitive Grants Program, and limited funding through CSREES to Regional Research Programs (e.g., NC-168) and a National Research Support Program, NRSP-8. NRSP-8 provides for coordination of animal genome research via the appointment of Species (Poultry, Cattle, Sheep, Swine) Technical Committees and Coordinators. Parallel developments in animal agriculture genomics have occurred world-wide, particularly in Europe (e.g., Institute for Animal Health, Compton, England; Roslin Institute, Edinburgh, Scotland; University of Wageningen, The Netherlands). One of the major goals of the NAGRP is to cooperate with international efforts to enhance progress in animal genetics.

anonymous viability genes, and neuropeptide/neurotransmitter genes).

Two major strategies are being used to locate and characterize genes of interest. In the candidate gene approach, potential ETL-associated genes are chosen based on the physiological or genetic effects of the proteins or RNAs they encode as studied either in poultry or other species. The candidate gene is then isolated by recombinant DNA techniques, further characterized, and used to generate markers to test its usefulness as an ETL indicator in appropriate populations. In the map-based approach, anonymous ETLs are mapped in a test or resource population. Then a gene that is closely associated with a particular ETL can be isolated based on its location in the map, or by a combination of candidate and map-based techniques. A fundamental tool required for the latter approach (and also useful for the candidate-gene approach) is a high quality, integrated genome map. High quality means densely spaced markers that are useful in many populations; integrated implies that the poultry genetic, cytogenetic, and physical maps are also correlated with genome maps of other animal species. The identification of conserved syntenic groups between poultry genomes and those of humans and mice will facilitate the use of genetic and physiological data from these more highly studied species.

A critical point with regard to genome research is the availability and suitability of poultry genetic resources. While commercial breeders maintain populations that presumably contain a wide variety of alleles, these populations are often not useful for research purposes if these breeders are unwilling to make populations (or data) available because of trade secrecy (proprietary) concerns, or the commercial lines are so outbred and therefore so heterogeneous at economic trait loci that it becomes impossible to measure specific effects at one or a reasonable number of candidate loci. Therefore, genetic mapping experiments require the availability of diverse populations of well-characterized, inbred, or partially inbred (or carefully random-bred) germplasm. Public access to genetic stocks and information is essential for the advancement of science. Thus, the growing number of patent requests on such material by private corporations is a concern, as markers identified by researchers supported by funding from private organizations, using stocks that are privately owned (as with recent gene mapping studies by the Groenen group (CROOIJMANS *et al.* 1996) may be restricted in their availability.

Developmental genetics

Chicken developmental mutations played a significant role in the early days of the science of genetics. Bateson's work at the turn of the century showed that different comb shapes in the chicken followed Mendelian inheritance patterns. This proved that Mendel's principles, though discovered in plants, also applied to animals (BATESON 1902; BATESON and PUNNETT 1906, 1908). Today, there is increasing interest in the molecular genetic analysis of such developmental or pattern mutants to determine the basis of the developmental defects that produce these changes. The defined physical defects associated with the avian developmental mutations, exemplified in Figures 11 and 12 and Box 17, contrast strongly with many of the synthetic (knock-out) mutations in the mouse, whose phenotype is often simply characterized by the timing of early embryonic death.

Currently, developmental genetics is one of the most rapidly advancing areas in the biologi-

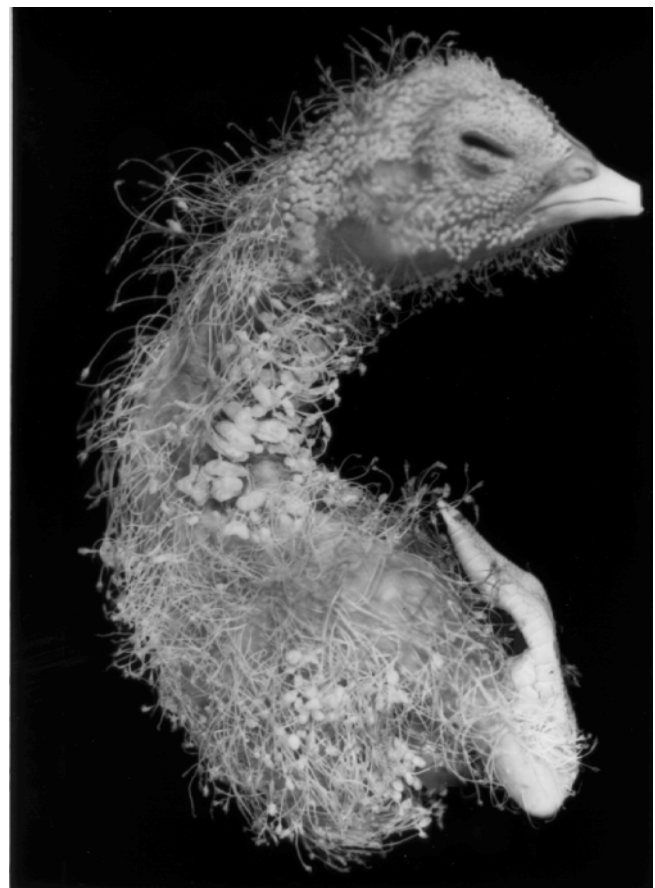


Figure 11. *Wingless-2* chicken embryo from UCD Wingless-2 X 331 (Photo courtesy of J.M. Pisenti, University of California–Davis).

cal sciences (RIDDLE *et al.* 1993; FALLON *et al.* 1994; MORGAN 1997; SANDERS and WRIDE 1997; CHUONG 1998). Questions that have intrigued and puzzled embryologists and geneticists for decades can finally be addressed. The award of the 1995 Nobel Prize in Medicine/Physiology to E.B. Lewis, C. Nusslein-Volhard, and E.F. Wieschaus reflects the worldwide recognition of the fundamental importance of research in developmental genetics. Their work in identifying fruit fly (*Drosophila melanogaster*) developmental mutants and determining the molecular basis for some of these mutations provided key information for identifying a whole class of pattern-altering genes (the homeobox-containing genes) found in such diverse organisms as the fruit fly, nematode, echinoderm, fish, chicken, mouse, and human.

Avian developmental geneticists interested in pattern formation have studied an array of morphological mutants, most of which are recognizable early in development. These mutants typically alter the beak, head, trunk (including the tail), limbs, and integument (skin, feathers, and scales) (ABBOTT 1967; ROMANOFF 1972; LANDAUER 1967, 1973; SAWYER and GOETINCK 1988). In many instances, as exemplified by the *wingless-2* mutation in Figure 11, the mutations have a pleiotropic effect (alter more than one embryonic structure), with combinations of defects involving the limbs, trunk, beak, feathers, heart, kidneys, and so on. Another tantalizing finding, much investigated by LANDAUER (1967, 1973), reflected the close similarity of many known mutant syndromes with those produced by treatments with a variety of chemicals or hormones, or caused by nutritional deficiencies or excesses



Figure 12. *Eudiplopodia* foot from UCD *Eudiplopodia* X 003 (Photo courtesy of J.M. Pisenti, University of California–Davis).

(the so-called phenocopy effect). A field with great potential then involved studies of the basis of phenocopies and of means of overcoming them. Later, studies with pesticides provoked additional interest, as many of these environmental toxins could also produce phenocopies of different mutant syndromes.

Currently, the genes affecting patterning in the avian embryo are increasingly important in developmental studies (RIDDLE *et al.* 1993;

Box 17. Patterns in vertebrate limb development

LIMBLESS IS AN AUTOSOMAL recessive mutation that causes the complete absence of limbs in homozygotes; heterozygotes have normal limbs (PRAHLAD *et al.* 1979). The earliest stages of limb development appear to occur normally in the mutant homozygotes, but shortly after the limb bud forms, it ceases to develop and soon regresses. Two recent studies of this mutation (GRIESHAMMER *et al.* 1996 and NORAMLY *et al.* 1996) have provided evidence that although the phenotype of the mutant is a lack of limb outgrowth, the primary defect in *limbless* embryos is a lack of normal dorsal-ventral patterning at a very early stage of limb development. This apparently results in an inability of the embryonic cells in the

prospective limb-forming territory to respond appropriately to the normal limb-inducing signal, and as a consequence the apical ectodermal ridge does not form. In the absence of this important signaling center, limb outgrowth does not occur. The importance of these studies is that they have revealed a previously unknown link between patterning along the dorsal-ventral axis and the initiation of limb bud formation.

This breakthrough in understanding the fundamental mechanism of limb development in vertebrates would not have been made were it not for the availability of chicken stocks carrying the *limbless* mutation, because similar mutations in other species such as mice and humans are not known. However, it is ex-

tremely important to bear in mind that the discovery that *limbless* encodes a gene playing a critical role in limb development could not have been made before the molecular markers for dorsal and ventral cell identities became available. Other mutations in avian stocks may be equally valuable, but their importance cannot be assessed at present because of limitations in our knowledge about limb development or because the appropriate markers for assaying gene function are not available. If we discard stocks because we do not appreciate their value, we may be discarding the key to a more profound understanding of a particular developmental or disease process.

RODRIGUEZ *et al.* 1996). Specifically, these mutations can be used to study signaling, biological clocks, interactions between different developmental controls, and the relationship of the defective patterns to similar syndromes produced by chemical or other experimental means. Importantly, these mutants can be used to investigate directly various hypothetical models of gene action developed by other means.

Limb development

For more than 50 years, vertebrate limb development has been a major focus of developmental biology (SAUNDERS 1948; ABBOTT 1967; SAWYER and GOETINCK 1988; FALLON *et al.* 1993; LAUFER *et al.* 1997). Between 1972 and 1998, advances in this field have been highlighted at six International Conferences on Limb Development and Regeneration. The limb bud has been of interest because it provides an experimental paradigm for exploring fundamental mechanisms of vertebrate tissue outgrowth and patterning (SAUNDERS 1972; EDE *et al.* 1977; HINCHLIFFE and JOHNSON 1980; CONNELLY *et al.* 1981), and for identifying the molecules that mediate these processes. It also provides a model for studying the mechanism of embryonic induction (CONNELLY *et al.* 1981). Most of what is presently known about vertebrate limb development has come from studies performed in avian species, primarily the chicken, but it is now clear that the mechanism of limb development has been highly conserved throughout evolution, and what has been learned about limb development in the chick is equally informative about limb formation in mammals (FALLON *et al.* 1993). Indeed, it appears that the early steps in limb development and the molecules that perform them are virtually identical in all vertebrate species (MORGAN 1997), and it is only at relatively late stages that species-specific differences become significant. A number of researchers have used developmental mutations to test hypothetical control mechanisms governing limb pattern at the tissue level. More recently, still other researchers have returned to

these mutations to study perturbations in the expression of developmentally regulated molecules that could explain the pattern abnormalities characteristic of such mutations (GRIESCHAMMER *et al.* 1996; NORAMLY *et al.* 1996; RODRIGUEZ *et al.* 1996; LAUFER *et al.* 1997). Thus, there is still much to be gained from the analysis of the mutations that have occurred in avian stocks and which have been maintained in university collections. Two particularly useful chicken mutations are the *limbless* mutation, which has recently been used to gain a remarkable insight into the fundamental mechanism of limb initiation (Box 17), and the *eudiplopodia* mutation (Figure 12), which has showcased the critical nature of timing and location of signaling molecules in early limb bud on the final patterning of the limb (LAUFER *et al.* 1997).

Feather and scale development

The morphogenesis of cutaneous appendages, such as feathers, scales, and hair, depends on a series of reciprocal interactions between the epithelial and mesenchymal layers of the embryonic skin (SENGEL 1976). Such epithelial-mesenchymal interactions also take place in the development of the limb (RIDDLE *et al.* 1993; FALLON *et al.* 1994; NISWANDER *et al.* 1994), tooth (VAINIO *et al.* 1993), kidney (PATTERSON and DRESSLER 1994), lung (PETERS *et al.* 1994), and mammary gland (CUNHA 1994). Evidence is accumulating which indicates that the epithelial-mesenchymal signaling that occurs during the early morphogenesis of these various organs relies on common molecular mechanisms (Box 18 and CHUONG 1998). Thus, information gained on the mechanisms for any one of these organs systems is very likely to be applicable to our understanding of patterning events in other systems. A future challenge is to understand how different embryonic structures acquire their identity while relying on common signaling mechanisms.

Box 18. Patterns in skin development

THE *SCALELESS* MUTATION has contributed significantly to our understanding of both the cellular and molecular mechanisms that control the formation of cutaneous appendages, particularly the feathers and scales in birds. Chickens homozygous for the *scaleless* mutation (Figure 13), an autosomal recessive trait, lack most feathers and scales (ABBOTT and ASMUNDSON 1957) as well as the scleral ossicles (the precursors to the bony eye ring) (PALMOSKI and GOETINCK 1970). These defects have made this mutant an ideal model system for studying the early development of cutaneous appendages, and, by extrapolation, other organ systems depending on epithelial-mesenchymal interactions. In some of the earliest studies in which normal and *scaleless* ectoderm and mesoderm were recombined, researchers showed that the *scaleless* mutation specifically affects the ectodermal epithelium (GOETINCK and ABBOTT 1963; SENDEL and ABBOTT 1963). This ectodermal defect prevents the formation of the ectodermal placodes in the skin of *scaleless* embryos (GOETINCK and SEKELICK 1972), thus interrupting the normal sequence of tissue interactions. Despite the ectodermal defect, the mutant mesenchyme is fully capable of participating in feather and scale development when recombined with genetically normal ectoderm (GOETINCK and ABBOTT 1963; SENDEL and ABBOTT 1963; SONG and SAWYER 1996).

Interestingly, the mutant phenotype can be greatly modified through selection for increased or decreased number of feathers. The accumulated modifier genes for increased feathering alter the expression of the *scaleless* gene by altering the response of the mesodermal signals (BROTMAN 1977a,b). A number of studies also addressed the cellular and subcellular (histological) differences in the devel-

opment of the feathers and scales (SAWYER and ABBOTT 1972; SAWYER *et al.* 1974; SAWYER 1979). These classical studies in the genetics, histology, and tissue and cell biology of this mutation laid essential groundwork for current molecular studies that were designed to explore molecular control mechanisms governing feather and scale development.

In a recent study, the role of fibroblast growth factor (FGF) signaling was examined in the epithelial-mesenchymal interactions during the initiation of feather germ formation in genetically normal and *scaleless* embryos. A spatially and temporally restricted pattern of transcription was seen for the genes that encode FGF-2 and FGFR-1 in developing feather germs of the normal chick embryo. FGF-2 expression is restricted to the early feather epidermal placodes, whereas, FGFR-1 expression is limited to the dermal condensations underlying the placodes. Transcription of these genes

could not be detected in skins of *scaleless* embryos that failed to develop feathers as a result of their ectodermal defect. However, when *scaleless* skin was treated with FGF-2, feathers would form at the site of application of the growth factor, and the induced feathers express FGFR-1 in their dermal condensations. However, the response was restricted to a narrow window of time, strongest around day seven and eight, and gone by day 11 (SONG and SAWYER

1996). Based on these observations, FGF-2 was hypothesized to be an active molecular component of the early signaling between the dermal mesenchyme and the overlying epithelium during feather morphogenesis. The observation that FGF-2 can rescue the mutant phenotype of *scaleless* embryos suggests that FGF-2 either is, or is downstream from, the signal that the ectoderm of the *scaleless* mutant fails to generate.

The results obtained with the *scaleless* skins indicate that this mutant is an excellent system to elucidate signaling pathways by FGF and other signaling molecules in the morphogenesis of cutaneous appendages. Mechanisms identified from this system may be applicable to the understanding of developmental mechanisms in other organs in which epithelial-mesenchymal interactions play a role (PETERS *et al.* 1994).



Figure 13. Hens from UCD Scaleless-Low (Photo courtesy of J. Clark, University of California–Davis).

Conserving avian genetic resources

CONSERVATION OF AVIAN WILDLIFE species receives global attention because of the large numbers of species presently in decline, and because birds provide a focus for recreational activities throughout the world. Less known are the needs for conservation of progenitor, landrace, and feral populations of domesticated birds. Even less well publicized is the critical situation of many of the specialized stocks used in research, as demonstrated in Chapter 5.

Safeguarding existing biodiversity is a common theme in the conservation of all types of avian genetic resources, but the strategies used to meet this goal can be quite different, depending on the species. Wild species are generally conserved through natural habitat protection and take regulations (i.e., hunting or harvesting limits for a given species), with captive breeding used in only the most severe cases of species decline. Measuring the genetic diversity in wild species is being addressed in a relatively few cases with DNA markers. Such data contribute to the development of rational schemes for conserving genetic diversity. Wild progenitors of domesticated birds, such as the Red Jungle Fowl are treated similarly, but wild turkey conservation, as another example, is enhanced by restocking and translocation of individuals. This activity is motivated by the need for conservation of the species and its diversity, but the fact that turkey is a recreational game bird is perhaps a stronger motivation for conservation.

The goal of genetic resource conservation is the maintenance of genetic integrity of a species or populations within a species. For populations in nature, natural evolutionary processes remain active, thus changes in gene frequency, population sizes, and geographic distribution are to be expected. Human interventions impact species composition through disruption of habitat and harvesting. In such cases, proactive conserva-

tion strategies must be applied. The situation is similar for domesticated breeds of birds, but there is more concern for conservation of specific combinations of traits and genes, with minimal changes in gene frequency. Even more restrictive is conservation of genetic stocks where specific genes are targeted. For these genetic entities, no change in gene or genotype frequency can be accepted. Thus, we recognize a distinction between conservation and preservation. The former allows utilization with minimal genetic changes over time caused by human activity and the latter requires that no genetic changes occur, exemplified by nonliving materials, quiescent (e.g., cryopreserved) specimens of gametes and embryos, or closely bred living genetic stocks.

Assays for DNA polymorphisms can be done with nonliving biological materials, providing the opportunity for assessment of genetic diversity in historical times from museum specimens or preserved tissues, including blood serum. This emphasizes the value of preserved biological materials as a component of a conservation strategy for birds. Cloned genomic or cDNA libraries can be preserved indefinitely at low temperatures. This includes DNA clones used for preparing molecular linkage maps and for discovery of functional genes. Databases of DNA nucleotide sequences of genes or expressed sequence tags are extremely rich sources of information because sequences from many species are available for screening and comparisons with unknown sequences.

This report focuses on the conservation of genetic stocks, as defined earlier. However, a holistic view of avian conservation is appropriate because the methods used for preserving genetic stocks and breeding populations, especially semen cryopreservation, can be adopted or modified for other species, including threatened or endangered wild species. Conservation methods

need to be adopted which meet the specific requirements of the species or the trait (genes) being conserved. An avian genetic resource conservation system includes several components (acquisition, maintenance, utilization, distribution, documentation, and health and quarantine) which can provide broader service than the conservation and distribution of genetic stocks. This is elaborated in Chapter 6.

Methods of conservation of genetic stocks

Avian genetic materials may be preserved in the form of live birds, as cryopreserved semen and dispersed embryonic or primordial germ line cells, as preserved tissues from which DNA can be isolated, as cloned genomic or cDNA, or as nucleotide sequence data. Of these methods, living birds and cryopreservation methods permit recovery of animals, while preserving DNA gives the potential of recovering selected genes or defined segments of chromosomes. However, it should be remembered that at best each DNA sequence represents only a very small portion of a total genotype and that, while samples of total DNA from a population may be preserved, sorting out the genes useful for a particular purpose may be a difficult task, particularly when gene interactions must be taken into consideration. Cryopreservation of semen and embryonic cells is gaining acceptance as a means of preserving endangered genetic resources, including wild species, rare breeds, and mutant or specialty lines used in research.

Ultimately, the preferred conservation method depends upon 1) the reliability of the method to yield the desired number of live animals, 2) the characteristics of the stock, and 3) the availability of technical support, instrumentation, and storage facilities. Ideally, more than one method of conservation should be used, or there should be a backup site to reduce the risk of loss due to fire, equipment failure, and human errors. Below are outlined some features of live-bird conservation and three methods of cryopreservation of semen or embryonic cells from which live birds may be recovered. Semen cryopreservation has the longest history, although not a highly efficient recovery rate. Dispersed blastodisc and primordial germ cell cryopreservation are promising emerging technologies that will require further research to improve recovery rates to more acceptable levels.

Live animals

The maintenance and periodic reproduction of live animals is the most dependable alternative available for preserving a genetic stock. This is particularly true for the inbred and long-term selected stocks, with their integrated collections of quantitative trait alleles. This is also the most reasonable way to maintain stocks that are often used in research. However, due to financial constraints, most researchers cannot justify the live maintenance of stocks that are used only sporadically, and recent events have shown (Chapter 5) that many unique lines are terminated for this reason.

The average number of birds required to maintain a given genetic stock depends upon its characteristics. Randombred and selected stocks require a larger base population (often several hundred birds) than those carrying single-gene mutations. The latter may be realistically continued with fewer than ten mutant-carrier birds if a robust outbred stock is used as one parent line each generation. Other factors that affect the number and distribution of birds needed to maintain a viable population include bird liveability, amount of inbreeding depression, endemic diseases, and the risk of natural or man-made disasters, which can literally wipe out genetic stocks in a very short period of time.

Maintaining stocks as live animals is very expensive, especially the selected lines and randombred populations. Even temporary shifts in interests of the curator or sponsoring institution can quickly orphan such genetic stocks, often leading to stock elimination and extinction of valuable genes or gene combinations. These realities are documented in Chapter 5, emphasizing the need for backup sites or a permanently dedicated conservation program.

Semen cryopreservation

The technology for semen cryopreservation (BAKST 1990; BUSS 1993; HAMMERSTEDT 1995) has been successfully adapted for the preservation and propagation of chickens and certain feral and endangered species (GEE 1995). Many different cryopreservation protocols have been evaluated (SEXTON 1979, LAKE 1986, HAMMERSTEDT and GRAHAM 1992, BUSS 1993, HAMMERSTEDT 1995). According to HAMMERSTEDT (1995) semen cryopreservation is the most cost-effective germplasm conservation strategy. Hammerstedt outlined the benefits of semen cryopreservation for the commercial poultry industry, but this preservation technique has not yet been widely

adopted. An important feature of semen cryopreservation for unique or endangered poultry lines is that semen can be collected frequently (twice weekly) from a small number of males for use when females become available or in the future establishment of founder flocks. Other benefits of semen cryopreservation are:

- Frozen semen is not easily affected by diseases or natural disasters.
- Semen cryopreservation from many birds of several lines can be accomplished at a relatively low cost
- The expense of maintaining a flock is reduced or eliminated.
- Frozen semen is more easily transported nationally or internationally than live birds or fertile eggs (HAMMERSTEDT 1995).

There are obvious limitations to the routine use of semen cryopreservation. For some species and genetic stocks the method may not be reliable or yet suitably developed. Another consideration is that only the male genome is preserved, which means that the complex genome of highly inbred or selected stocks could not be preserved intact if only maintained as frozen semen, because, of course, none of the corresponding inbred or selected females would be available for insemination with the cryopreserved material. Thus, while semen cryopreservation permits the incorporation of preserved gene complexes into diploid animals, these gene complexes would be heterozygous in the progeny and subject to genetic recombination in subsequent intermatings of progeny. On the positive side, semen cryopreservation may prove to be the most appropriate method for preserving single-gene mutant stocks, particularly if the genetic background is not a great concern in the modification of the mutant gene expression.

Another problem with cryopreserved semen is that the quality of thawed semen is currently quite low. This is reflected in fertility levels rarely exceeding 70%, and more typically less than 50%, due to sperm destruction and deformity caused by semen cryopreservation and recovery procedures. One researcher estimated that cryopreserved semen has less than 2% of the fecundity of fresh semen (WISHART 1985). This is in spite of the fact that most of the successful cryoprotectants, diluents, and freezing techniques were developed with semen from highly productive commercial broiler chicken stocks.

Unfortunately, the semen from inbred, specialty, and mutant-carrier stocks most at risk often does not respond well to cryopreservation procedures that worked reasonably well with commercial stocks. Frozen-thawed semen from these less-robust stocks often exhibit extremely high or complete infertility associated with poor motility and high numbers of freeze-damaged or killed spermatozoa. Obviously, further research is needed, not only in the development of improved semen cryopreservation procedures (improved diluents, cryoprotectants, and other supplements, and optimizing cooling and thawing rates), but also addressing the significance of species variation, male-line variation within a species, and individual male variation within a given strain. However, despite the potential utility of this procedure in preserving the germplasm of rare or endangered stocks or species, only a few researchers in Japan, England, and the US are currently conducting research on semen cryopreservation.

Blastodisc cryopreservation

Cryopreservation of the cells from an avian blastodisc (Figure 14, the embryonic cells in an unincubated fertile egg) is a new technique that can be used to preserve the intact genome of poultry genetic stocks (REEDY *et al.* 1995, Box 19). While the methods are still being perfected, frozen-thawed blastodisc cells have been successfully integrated into the blastodiscs of host chicken eggs, and developed into reproductively competent adults. The long-term viability of cryopreserved blastodisc cells is not yet known, although cryopreservation studies with semen

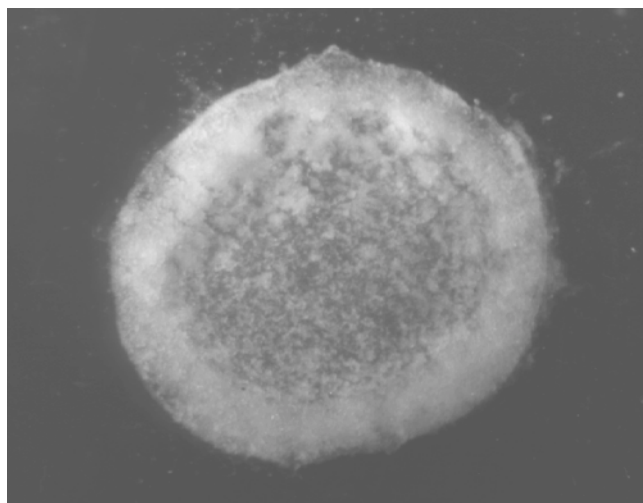


Figure 14. Stage X blastodisc (at time of oviposition) (Photo courtesy of M.E. Delany, University of California-Davis).

and other cell-types suggests that properly cryoprotected and frozen specimens will remain viable indefinitely if kept in liquid nitrogen.

The large size and complex structure of the avian embryo (already composed of 40,000 to 50,000 cells when the egg is laid) do not permit the application of techniques that have been developed for cryopreservation of mammalian embryos. A different strategy is required which uses dispersed cells isolated from the relatively undifferentiated embryonic cells present in the newly laid fertile egg (Box 19). Recent experiments have demonstrated that frozen-thawed blastodermal cells from purebred chickens of the Barred Plymouth Rock breed can successfully integrate into an irradiated White Leghorn host blastoderm to form embryos that are somatic

and germline chimeras. These chimeric chickens hatch and mature normally, and, when interbred, produce some offspring that are purebred Barred Rock (KINO *et al.* 1997). While frozen-thawed blastodermal cells form germline chimeras less frequently than fresh blastodermal cells, a sufficient number of “reconstituted” birds of lines maintained as cryopreserved blastodermal cells can be obtained to use the technology to store valuable genetic material (KINO *et al.* 1997). Thus, this technique should eventually provide an inexpensive long-term solution for preserving the intact genome of stocks (particularly inbred and selected stocks) that are considered to be valuable genetic resources but are not currently required in an active research program (Box 20).

Box 19. Mosaic chickens and genetic stock preservation

A NEW TECHNIQUE HAS BEEN developed at the University of Guelph (Ontario, Canada) that can be used to reclaim both freshly dissociated and cryopreserved chicken embryonic cells (Figure 15, REEDY *et al.* 1995). Briefly, cells from the stage X embryo (EYAL-GILADI and KOCHAU 1976) can be removed, dispersed, and injected into recipient blastodiscs that are at the same stage of development. The resulting chimeras (mixture of host and donor cells), which can be either male or female, produce donor-derived gametes at rates of up to 100% (PETITTE *et al.* 1990; 1993; CARSIENCE *et al.* 1993; THORAVALL *et al.* 1994; KAGAMI *et al.* 1995). Matings of male and female chimeras that each produce donor-derived gametes yield offspring with the complete donor genotype at a rate that is the product of the frequency of the production of donor-derived gametes by each of the chimeric parents (KINO *et al.* 1997).

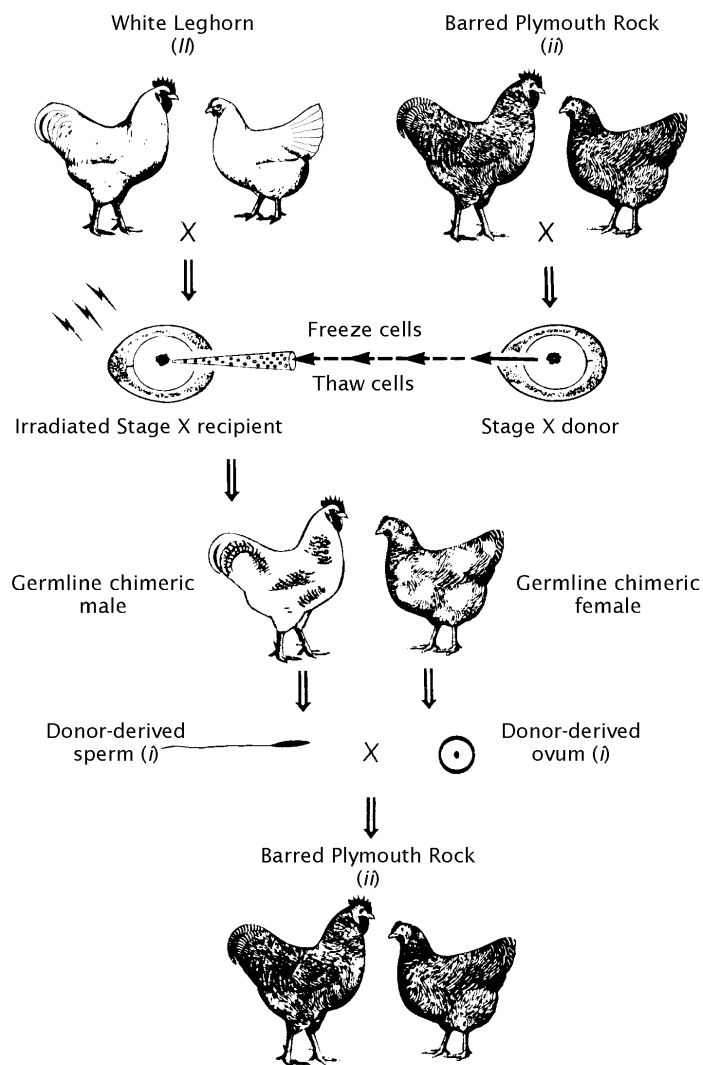


Figure 15. Diagram of avian blastodisc cryopreservation and recovery (from REEDY *et al.* 1995).

Research aimed at improving the rate of germline transmission by cryopreserved blastodermal cells could increase the effectiveness of this technology and reduce the number of blastoderms that are required to ensure that the genetic diversity within a stock is maintained in the reconstituted population. While this would be a welcome improvement in the technology, it should be emphasized that the current technology can be used to conserve genetic resources and that it is the only fiscally realistic way to maintain the genome of stocks in an unadulterated state at the present time.

In the future, the range of genetic diversity may increase as transgenic stocks are produced. Many of these stocks will be developed for specific experimental uses that have short-term utility in research. It would be prudent to store these stocks as frozen blastodermal cells rather than discard the time and effort expended in their production.

Primordial germ cell cryopreservation

Cryopreservation of primordial germ cells (PGCs) isolated from the blood of early-stage chick embryos is another way of preserving the intact genome of both male and female birds (NAITO *et al.* 1994a). The PGCs are the precursors of the adult gametes (ova and spermatozoa) and form a distinct population of readily distinguishable cells even before they move from the extra-

Box 20. Orphaned CFAR stocks preserved as frozen embryonic cells

THE FIRST LARGE-SCALE DEPOSITION of frozen blastodermal cells was completed in 1998, preserving over 30 of the stocks once kept at the Centre for Food Animal Research (CFAR). This had been the flagship research center for poultry research for Agriculture and Agri-Foods Canada. The cryopreserved CFAR stocks were among those that could not be placed with other institutions before April 1, 1997. Blastoder-

mal cells were harvested and frozen at the Avian Physiology and Genetics Laboratory at the University of Guelph, (Ontario, Canada). If these stocks are required in the future, trained technicians can thaw the cells and make germline chimeras (see Box 19), which can then be mated to obtain pure "reconstituted" birds from the original CFAR lines.

embryonic germinal crescent into the blood vessels. After a short period of time being carried through embryonic and extra-embryonic circulatory systems, these cells will migrate out of the blood vessels and selectively colonize the gonadal ridges. While in the migratory phase (mostly between HAMBURGER and HAMILTON (1951) stages 13 and 15), the PGCs can be removed from the embryo in blood samples. Primordial germ cells can be partially separated from the blood cells by density gradient centrifugation and can then be cultured (CHANG *et al.* 1995; KUWANA *et al.* 1996), cryopreserved (NAITO *et al.* 1994a), or immediately injected into a host embryo (TAJIMA *et al.* 1993; NAITO *et al.* 1994b). One of the benefits of this method of germplasm preservation is that a highly enriched population of germline cells of approximately 60% PGCs (NAITO *et al.* 1994b) can be introduced into a host embryo. Thus, even when frozen-thawed PGCs were injected, up to 25% of the gametes were shown to be from the donor germline (NAITO *et al.* 1994a). This germline recovery rate is substantially better than that obtained from frozen-thawed blastodisc cells (KINO *et al.* 1997).

Genetic stocks: Current resources and recent losses

ALMOST ALL OF THE GENETIC STOCKS of chicken, turkey, Japanese quail, and other domesticated birds that have been used in research in the US and Canada were developed and conserved by public sector universities and federal government research organizations in these two countries. Many in the research community have been aware of the gradual attrition, and occasional dramatic losses, of these specialized stocks over the years. Some important genetic resources were discarded before relocation plans could be developed or because a new stock curator could not be located. Current threats to additional abandonment of existing genetic stocks gave impetus for a study of the true situation of existing genetic stocks, where such stocks are located, and their status for conservation and use. The situation is dynamic, for not only are established genetic resources at risk, but new genetic stocks are also being developed in current research programs that will in turn require preservation or conservation in the years to come. SOMES (1988) provided a benchmark with his comprehensive, but not exhaustive, summary of the state of genetic stocks up to 1988. Hence the Task Force undertook an assessment of the current situation with the goal of producing a complete database inventory of extant genetic stocks, along with a summary of the genetic stocks that had been discontinued, abandoned, or lost since 1984. This information constitutes the basis for the proposal of a comprehensive strategy geared towards the conservation of avian genetic stocks important to basic biological research and education. (See Chapter 6).

Survey methods

The study was conducted by Jacqueline Pisenti during 1995–97 at the UC Genetic Resources Conservation Program, with additional financial

support from the National Science Foundation and the USDA Agricultural Research Service. In 1998 each curator was contacted again to produce an updated report. A survey instrument (Appendix 1) was sent to all of the curators mentioned in SOMES (1988) and the participants in the CSREES Regional Research Project NE-60 on genetic basis for resistance and immunity to avian diseases. Individuals listed in the Poultry Science Association 1995–96 membership directory who indicated a focus on genetics or immunology, and individuals on the 1997 Poultry Science Resource List prepared by the Poultry Science Association and R.D. Reynells (USDA/CSREES) were also contacted (usually via email or telephone) and queried. In all, survey forms were sent to 37 curators (11 in Canada and 26 in the US) at 27 institutions listed in the SOMES (1988) registry and 72 additional individuals were contacted by phone or email. All curators were asked to report on the status of stocks that had been included in the 1988 registry (i.e., which of these stocks had been retained, transferred, or eliminated), and to list any new stocks acquired since 1988. They were also asked to indicate the prognosis for long-term security of each stock. Finally, respondents were asked to send the names of any poultry genetic stock curators they were aware of, either at their institutions or elsewhere, and these individuals not already contacted were also asked to respond to the survey.

Results from the survey

According to the survey, 361 genetic stocks are currently maintained (Table 1 and Appendix 2). Of the 36 curators at 27 institutions listed in the 1988 registry (SOMES 1988), 31 of them (or their successors) responded; four no longer maintained stocks, and 27 still had at least some of

the stocks listed in the 1988 registry. Of the 72 other persons queried, 44 responded and 13 reported that they curated poultry genetic stocks.

By species, there are 268 chicken stocks (with 38 existing only as cryopreserved material), 65 Japanese quail, 20 turkey, six waterfowl, and two gamebird. Table 1 shows the categorization

of the existing genetic stocks. The detailed database in Appendix 2, Table 2.1, lists the characteristics of every genetic stock that was identified in the survey. These were grouped into ten main categories. The largest category is single-gene mutants, with 107 stocks that display color variations or defects in developmental, immunologi-

Table 1. Summary by category of existing poultry genetic stocks (live and cryopreserved).

Category	Chicken	Japanese quail	Turkey	Waterfowl, Gamebird	Total
Number of stocks					
Bloodtype-Gene pool	6	0	0	2	8
Bloodtype-Major Histocompatibility Complex (MHC)	13	0	0	0	13
Bloodtype-MHC-Inbred	51	0	0	0	51
<i>subtotal</i>	<i>70</i>	<i>0</i>	<i>0</i>	<i>2</i>	<i>72</i>
Chromosomal variant	5	0	0	0	5
Endogenous virus	1	0	0	0	1
Endogenous virus-Inbred	4	0	0	0	4
<i>subtotal</i>	<i>5</i>	<i>0</i>	<i>0</i>	<i>0</i>	<i>5</i>
Inbred	27	1	0	0	28
Mutant-Color, eggshell	0	2	0	0	2
Mutant-Color, feather	1	12	0	0	13
Mutant-Developmental defect-Eye	5	0	1	0	6
Mutant-Developmental defect-Face/limb	28	0	0	0	28
Mutant-Developmental defect-Skin/feather	8	6	0	0	14
Mutant-Developmental defect-Spine/tail	3	0	0	0	3
Mutant-Gene pool	7	1	0	0	8
Mutant-Immunological defect	5	0	0	0	5
Mutant-Neurological defect	3	0	0	0	3
Mutant-Physiological defect	12	0	0	0	12
Mutant-Reproductive defect	3	0	1	0	4
Mutant-Uncategorized	8	1	0	0	9
<i>subtotal</i>	<i>83</i>	<i>22</i>	<i>2</i>	<i>0</i>	<i>107</i>
Pure breed	12	0	5	6	23
Ranombred	18	14	8	0	40
Selected-Behavioral trait	2	1	0	0	3
Selected-Egg trait	11	0	2	0	13
Selected-Growth trait	14	18	3	0	35
Selected-Immune trait	11	1	0	0	12
Selected-Physiological trait	2	8	0	0	10
<i>subtotal</i>	<i>40</i>	<i>28</i>	<i>5</i>	<i>0</i>	<i>73</i>
Transgenic	2	0	0	0	2
Uncategorized	6	0	0	0	6
Total	268	65	20	8	361

cal, neurological, physiological, or reproductive characteristics. The selected stocks category, with 73 stocks, are products of long-term selective breeding and are characterized by above- or below-normal performance in defined behavioral, growth, egg production, immunological, or physiological traits. The next largest category with 72 stocks consists of the bloodtype variants, representing most of the known forms of chicken erythrocyte and leukocyte alloantigens. The inbred category, with 28 stocks, consists of stocks approaching genetic homogeneity as a result of crossing full sibs or other close relatives for many generations. In addition to the inbred category, highly inbred stocks can be found in the bloodtype-MHC-inbred and endogenous virus-inbred categories. A few highly inbred stocks are also found among the mutant-developmental defect-face/limb category. The randombred category with 40 stocks and the pure breed category with 23 stocks are important as control or reference populations and represent gene pools from which many of the selected and mutant stocks were derived. The uncategorized group with six stocks are those stocks for which no description was available.

Forty-one researchers at 27 institutions (24 in the US and 3 in Canada) maintain 323 genetic stocks as living birds (Table 2). Six of these institutions maintain 20 or more stocks (University of Arkansas, University of British Columbia-Vancouver, University of Connecticut-Storrs, University of California-Davis, University of Wisconsin-Madison, USDA/ADOL East Lansing, Michigan). Six maintained between ten and 19, four had six to nine, and 11 had five or fewer genetic stocks. Two institutions in the US and four in Canada no longer keep such stocks.

In addition to the living bird collections, 87 genetic stocks are maintained under semen or blastodisc cryopreservation at five institutions. Such frozen reserves are kept at the University of Guelph (Guelph, Ontario) (33), the University of California-Davis (31), the USDA Avian Disease and Oncology Laboratory at East Lansing, Michigan (17), Pennsylvania State University (4), and the University of Wisconsin-Madison (2). Five of the cryopreserved stocks at University of California-Davis are no longer maintained as live birds. The 33 cryopreserved stocks at the University of Guelph are from the Canadian Food Animal Research collection and also are no longer maintained as live birds. Thus, 51 stocks maintained as live birds are also backed up by cryopreservation, while 38 stocks now exist only under cryopreservation.

At the time this report went to press, approximately one-third of the stocks kept as live birds were considered by their curators to be at high risk of elimination within the next few years (30% of the chicken, 35% of the turkey, and 49% of the Japanese quail stocks). Just over 40% were considered to be in a secure situation, including 42% of the chicken, 45% of the turkey, and 37% of the Japanese quail stocks. The remainder of the stocks (about 30%) were considered to be only moderately at risk.

Lost stocks

In the last 15 years, some 238 avian genetic stocks were reported as lost or eliminated by research institutions in the US and Canada (Table 3; this also includes those few stocks reported as transferred to private individuals or organizations). The actual number of lost stocks is probably much higher. The data in Table 1 show the numbers and percentages of genetic stocks lost, based on the current number of existing stocks and documented numbers of lost stocks. Some new stocks have been developed since the last survey, specifically in the MHC-defined endogenous virus and selected categories. These recent additions inflate the total numbers of existing stocks and bias downwards the estimated percentage of stocks lost. Even so, stock losses have been substantial: close to 40% of the reported living US stocks and over 60% of the Canadian stocks were discontinued since 1984, 42% of all stocks (Table 2). Most notable was the discontinuation of more than 30 genetic stocks by Agriculture and Agri-Food Canada at its facility in Ottawa. This clearly reflects a disturbing trend in avian genetic resources conservation.

Of all lost stocks, 75% were chickens, 11% were turkeys, 11% were Japanese quail, and 4% were gamebirds or waterfowl. Ironically, more than one-third of these stocks were reported lost between 1995 and 1998 while this survey was in progress, including 50 chicken stocks, 19 turkey stocks, 10 Japanese quail stocks, and four of the waterfowl stocks. The losses were heaviest (35% of all lost stocks) among the mutant and chromosomal variant categories, although the selected (25%) and the bloodtype-MHC-inbred (21%) categories were not far behind. Some of these lost stocks, such as the bloodtype-MHC specialized stocks, included genes that can be recovered by breeding and selection from existing genetic resources, but at a great cost of money and time. The now-extinct lethal and

Table 2. Number of poultry genetic stocks kept as live birds in institutions in the USA and Canada in 1998 and the number lost since 1984.

Curating institution	Kept as living birds [†]					Lost since 1984 [†]						% all [‡]
	C	JQ	T	W,G	Total	C	JQ	T	W,G	Total		
USA	Number of stocks											
Alabama: Auburn University	10	0	0	0	10	17	0	0	0	17	63	
Arkansas: University of Arkansas	12	9	0	0	21	1	2	0	0	3	13	
Arizona: University of Arizona	0	0	0	0	0	1	0	0	0	1	100	
California: University of California	46	1	0	0	47	15	7	1	4G	27	36	
Connecticut: University of Connecticut	20	0	0	0	20	8	1	1	0	9	31	
Delaware: University of Delaware	1	0	0	0	1	0	0	0	0	0	0	
Georgia: University of Georgia	4	9	0	0	13	2	6	0	0	8	38	
Illinois: Northern Illinois University	7	0	0	2G	9	0	0	0	0	0	0	
Illinois: University of Illinois	2	0	0	0	2	0	0	0	0	0	0	
Indiana: Purdue University	2	1	0	0	3	6	0	0	0	6	67	
Iowa: Iowa State University	17	0	0	0	17	5	0	0	0	5	23	
Iowa: USDA National Animal Disease Center	0	0	1	0	1	0	0	0	0	0	0	
Louisiana: Louisiana State University	0	3	0	0	3	2	0	0	0	2	40	
Maryland: University of Maryland	0	1	0	0	1	0	0	0	0	0	0	
Massachusetts: University of Massachusetts	4	0	1	0	5	10	0	1	0	11	69	
Michigan: USDA Avian Disease & Oncology Lab.	37	0	0	1W	38	2	0	0	0	2	5	
Minnesota: University of Minnesota	0	0	0	0	0	5	0	0	0	5	100	
Nebraska: University of Nebraska	1	1	0	0	2	0	0	0	0	0	0	
New Hampshire: University of New Hampshire	14	0	0	0	14	14	0	0	0	14	50	
New York: Cornell University	7	0	0	1W	8	7	1	0	0	8	50	
North Carolina: North Carolina State University	5	0	7	2W	14	0	0	2	0	2	13	
Ohio: Ohio State University	2	7	6	0	15	15	0	2	0	17	53	
Oregon: Oregon State University	1	0	0	0	1	0	0	17	0	17	94	
Pennsylvania: Pennsylvania State University	6	0	0	0	6	0	0	0	0	0	0	
Virginia: Virginia Polytechnic Inst. & State Univ.	4	0	0	0	4	0	3	0	0	3	43	
Wisconsin: University of Wisconsin	19	1	3	0	23	6	0	0	0	6	21	
Total in USA	221	33	18	4W 2G	278	116	20	23	4G	163	37	
Canada												
British Columbia: Univ. of British Columbia	3	32	0	0	35	0	5	0	0	5	13	
Ontario: University of Guelph	3	0	1	0	4	10	0	0	0	10	71	
Ontario: AA-FC Center for Food Animal Research	0	0	0	0	0	30	0	0	6W	36	100	
Quebec: McGill University	0	0	0	0	0	4	0	0	0	4	100	
Quebec: University of Lavale	0	0	0	0	0	1	0	0	0	1	100	
Quebec: Deschambault Research Station	0	0	0	0	0	4	0	3	0	7	100	
Saskatchewan: University of Saskatchewan	3	0	1	2W	6	11	0	1	0	12	67	
Total in Canada	9	32	2	2W	45	60	5	4	6W	75	63	
Total	230	65	20	6W 2G	323	176	25	27	6W 4G	238	42	

[†]C = chicken, JQ = Japanese quail, T = turkeys, W = waterfowl, G = gamebird.

[‡]“% all” indicates the proportion of total stocks at that institution that were lost since 1984.

subvital mutant stocks, along with the large number of discontinued translocation stocks, appear rarely, are difficult to detect, and are often discovered serendipitously. A number of the viable mutations that were kept in gene pools or as specialized stocks in research collections can still be found among hobbyist breeds or exhibition stocks, but they present problems in use because there is a risk of transferring diseases or they have background genotypes that may be inappropriate for use in research.

Individual stocks and whole collections that have been lost in recent years include:

- Inbred, specialty, and historical commercial stocks once maintained by Agriculture and Agri-Food Canada in Ottawa that were dispersed or eliminated in 1997, due to loss of government support. While some of these specialized stocks were sent to other institutions or private companies, 33 were retained only as cryopreserved blastodisc cells kept at the University of Guelph, (Ontario, Canada) (Box 20) and 30 were eliminated. However, one-half of these 30 were bloodtype stocks that are still available elsewhere.
- Five stocks (long-term inbred lines and one gene pool of dominant mutations) maintained at the University of Minnesota, were eliminated in 1996, although two (the dominant marker stock and the inbred Rhode Island Reds) had been transferred to the University of British Columbia a few years earlier.
- The collection of stocks at the University of Massachusetts went into dispersal in 1997, due to the retirement of its curator. While several important mutant stocks have found new curators, including the auto-immune vitiligo (DAM) chickens and their normal controls, the feather color and comb-type gene pool and tester stocks have been eliminated.
- At Oregon State University (Corvallis), a large and unique collection of 17 stocks carrying feather-color and embryo-lethal turkey mutations was eliminated in 1995, due to funding difficulties. A similar problem contributed to the loss of another collection of turkey stocks in the 1960s, this one held at the University of California-Davis.
- Fires at bird-care facilities have killed off at least two turkey stocks at the Deschambault Research Station and one Japanese quail mutation at University of California-Davis. Five other quail mutations salvaged from that fire at Davis (three unique to University of California-Davis) were subsequently eliminated due to funding difficulties.
- At the University of California-Davis, the retirement of two researchers with large collections of research stocks has put over 40 stocks in jeopardy (Box 3); these include over 20 mutations and an array of MHC-defined inbred lines, many of which are found nowhere else.

Table 3. Summary by category and species of poultry genetic stocks reported lost since 1984.

Category	Chicken		Japanese quail		Turkey		Waterfowl, Gamebird [†]		Total	
	No.	% [‡]	No.	% [‡]	No.	% [‡]	No.	% [‡]	No.	% [‡]
Bloodtype-MHC-Inbred	49	28	0	0	0	0	0	0	49	21
Chromosomal variant	33	19	0	0	0	0	0	0	33	19
Inbred	6	3	0	0	0	0	3G	30	9	4
Mutant	25	14	11	42	19	70	0	0	55	23
Pure breed	18	10	0	0	3	11	4W	40	25	10
Randombred	3	2	3	12	0	0	1W	10	7	4
Selected	42	24	11	46	5	19	1W,1G	20	60	5
Total	176	74[§]	25	11[§]	27	11[§]	6W,4G	4[§]	238	100[§]

[†]W=waterfowl; G=gamebird.

[‡]This column indicates the percentage of lost stocks in a category out of the total of lost stocks for that species.

[§]This value is the percentage of total lost stocks over all species accounted for by the total lost stocks for a given species.

Trends in poultry genetic stock development and conservation

Only a few large collections of vertebrate genetic stocks have been assembled, including the mouse (*Mus musculus*) collection that is currently kept at the Jackson Laboratory in Bar Harbor, Maine (Box 21) and the Japanese quail collection at the University of British Columbia (Box 22). But it is the chicken mutant collections that probably have the longest history, starting in the 1920s with Landauer and Dunn at the University of Connecticut, Storrs Agricultural Experiment Station (a collection that still exists, in part). These early researchers in vertebrate embryology used classical embryological techniques to study mutant gene expression, the effect of background genes on mutant phenotype, and the production of environmentally induced phenocopies (genetically normal embryos that resemble mutants) of specific mutant syndromes (LANDAUER 1973). Ironically, it is these areas of study that attract the attention of researchers today, just as the remaining academic avian genetic stock collections have become threatened. This underscores the need for preserving these unique gene pools and identified single-gene mutations for the benefit of future researchers. Similar arguments can be made for other research stocks, particularly inbred and selected strains. Both require many years and large, pedigreed populations during their development; factors that place formidable barriers to the regeneration of such stocks because of short-term and limited funding commitments.

Traditionally, poultry stocks were maintained at the research institutions where they were developed and studied. These institutions, frequently in states with a large or growing poultry industry, often had poultry or avian science departments that were independent of the larger animal science departments. This served to encourage research on the different poultry species and the development and maintenance of unique poultry genetic stocks. In the early 1960s, some 45 poultry science departments existed in the US; since that time and in spite of the tremendous growth in economic value of the the poultry industry and great consumer interest in poultry products, most departments have

been eliminated or merged with other departments, leaving only eight poultry science departments today (PARDUE 1997; DELANY and PISENTI 1998). With reorganization of these departments and their resources, genetic stock losses were inevitable as a result of loss of long-term support funds, reallocation of funding and resources to other areas, condemnation of poultry housing facilities without construction of adequate replacement housing, retirement of researchers who served as curator of stocks, shifts in research interests, and new research on exotic avian species as research models or companion birds.

The number of genetic stocks reported by SOMES (1988) is nearly the same as that detected in the present survey. However, the present survey documented the loss of nearly 200 chicken stocks, 23 turkey stocks, and at least 8 Japanese quail stocks, so it is evident that a considerable number of new stocks have been produced during the past decade, possibly displacing some of the older ones. Most curators faced with the need to reduce or disperse inventory try to find alternate conservators for stocks that are slated for elimination (Boxes 2, 3, 4), although this is not always successful (Box 8). Informal networking for new curators has worked well enough with single-gene mutation stocks when curating researchers had funding to support a number of these unique stocks, but this is often no longer an option.

Clearly, planned and unplanned stock losses are impacting the availability of specialized genetic resources to researchers. Stock eliminations are increasingly driven by funding problems, more will be lost. Particularly at risk are the selected lines and mutant stocks, which require either larger numbers or special handling to propagate.

Box 21. The Jackson Laboratory model for vertebrate genetic stocks

A GOOD EXAMPLE OF A SUCCESSFUL genetic stocks conservation program is the Jackson Laboratory, a non-profit, independent research institution, in Bar Harbor, Maine. Along with research and training, its third mission is the maintenance and distribution of a huge inventory of mouse genetic stocks. They are maintained as either live animals or frozen embryos and are distributed either as live animals or DNA preparations. Annually, the Laboratory supplies about two million mice from over 1,700 stocks to universities, medical schools, and research laboratories worldwide. In addition to providing the genetic stocks, the Jackson Laboratory also maintains and serves a computerized database system available anywhere in the world that enables fast, efficient access to a single comprehensive archive on the mouse. Supported partly by government grants and partly by user fees, the Jackson Laboratory is a good model for long-term preservation of avian genetic stocks. Further information on the Laboratory and the databases can be accessed from their web site (URL: <http://www.jax.org/>).

Box 22. Japanese quail at the University of British Columbia

PRIOR TO 1980, A JAPANESE quail colony was maintained at the Department of Animal Science, University of British Columbia (UBC) for research and teaching in genetics. In 1980, the goal was set to expand this colony and manage it as a genetic resource collection, and to make it available to users outside the department. In 1982, the Quail Genetic Resource Centre was formally established with the support of the department and the Natural Sciences and Engineering Research Council of Canada (NSERC). In the last 14 years, many new mutations and strains have been incorporated into the collection. At present, the quail populations at the Centre harbor 27 morphological and physiological mutations, close to 50% of all the mutations (not including isozyme variants) ever reported in the literature for this species. Detailed descriptions of the mode of inheritance, the phenotype, and possible applications of these mutations can be found in CHENG and KIMURA (1990). In addition, they also maintain 12 random-bred populations and specially developed strains that are useful for biological research or as breeding stocks for meat and egg production. Stocks from

the Centre have been widely utilized for research, teaching, and extension.

Areas of research include genetics, physiology, developmental biology, cancer, atherosclerosis, eye defects, neurology, animal behavior, and animal ecology. In the past three years, the Centre assisted or collaborated in more than 35 research projects. Currently, there are two on-going research projects using quail as a model to study atherosclerosis and another project using quail to study age-related macular degeneration (AMD). Except for monkeys, the quail is the only suitable model to study this disease which is the most common cause of blindness in humans over the age of 65. The Centre has also started screening chicken microsatellite DNA primers obtained from Hans Cheng to detect polymorphism in quail.

Teaching uses include courses in biology, genetics, poultry management, and animal behavior at University of British Columbia and other universities and colleges in the province. These stocks have also been used by students from local secondary schools for their senior science projects. The Centre staff also provides consultation for researchers and teachers on managing quail as a labora-

tory animal, and consultation for local producers on management of the flocks and/or marketing. In the latter cases, birds from the Centre were sometimes provided as breeding stocks. With the increase in consumption of quail products in metropolitan areas in Canada and also in the southern US, there has been a significant increase in requests for information from other parts of Canada and from the US.

The Centre has been partially supported by an infrastructure grant from NSERC, which provided the salary for the full-time technician. This grant covered 25% of the operating costs. Besides capital investments, the University provided 40% of the operating costs through department funds. The remaining 35% of the operating costs were covered by income from sales of products and users' fees. The NSERC support was terminated in 1995, and income generated from egg sales and research contracts are not sufficient to sustain the operation. An endowment will be sought to generate Canada \$30,000 Cdn a year to keep the operation going.

An Avian Genetic Resources System: Proposal

Task Force recommendation

IT HAS BECOME ABUNDANTLY CLEAR from the analysis of extant genetic stock collections and from the information obtained from task force members and others that many avian genetic stocks have been lost and many others are at serious risk of being lost. Further, the prognosis for maintaining new research stocks as they are being developed in current research programs is not good. In fact, when maintenance facilities for research stocks are not available, there will be no incentive for investment in lines of research that would produce such stocks. An avian genetic resources management system, with strong leadership, but shared responsibility, is proposed as the most efficient and secure way to conserve genetic stocks and address the concerns raised in this evaluation effort. The proposed Avian Genetic Resources System (AVGRS) would be comprehensive and would require the cooperation and collaboration of scientists, funding agencies, and research institutions. The System must be oriented toward research objectives, but it could also support the needs of breeders, breed hobbyists, and breed historians.

Rationale

Historic long-term collaboration between Canadian and US scientists provides a basis for collaboration in the formation and operation of an Avian Genetic Resources System. No comprehensive system exists in North America, nor anywhere else for that matter, for the conservation and distribution of avian genetic stocks (primarily chicken, turkey, and Japanese quail) of value for agricultural, biomedical, or biological research. The US National Plant Germplasm System and its counterpart in Canada serve as excellent models for the proposed Avian Genetic

Resources System. A System on this binational level is necessary because no dependable regional or local solution exists.

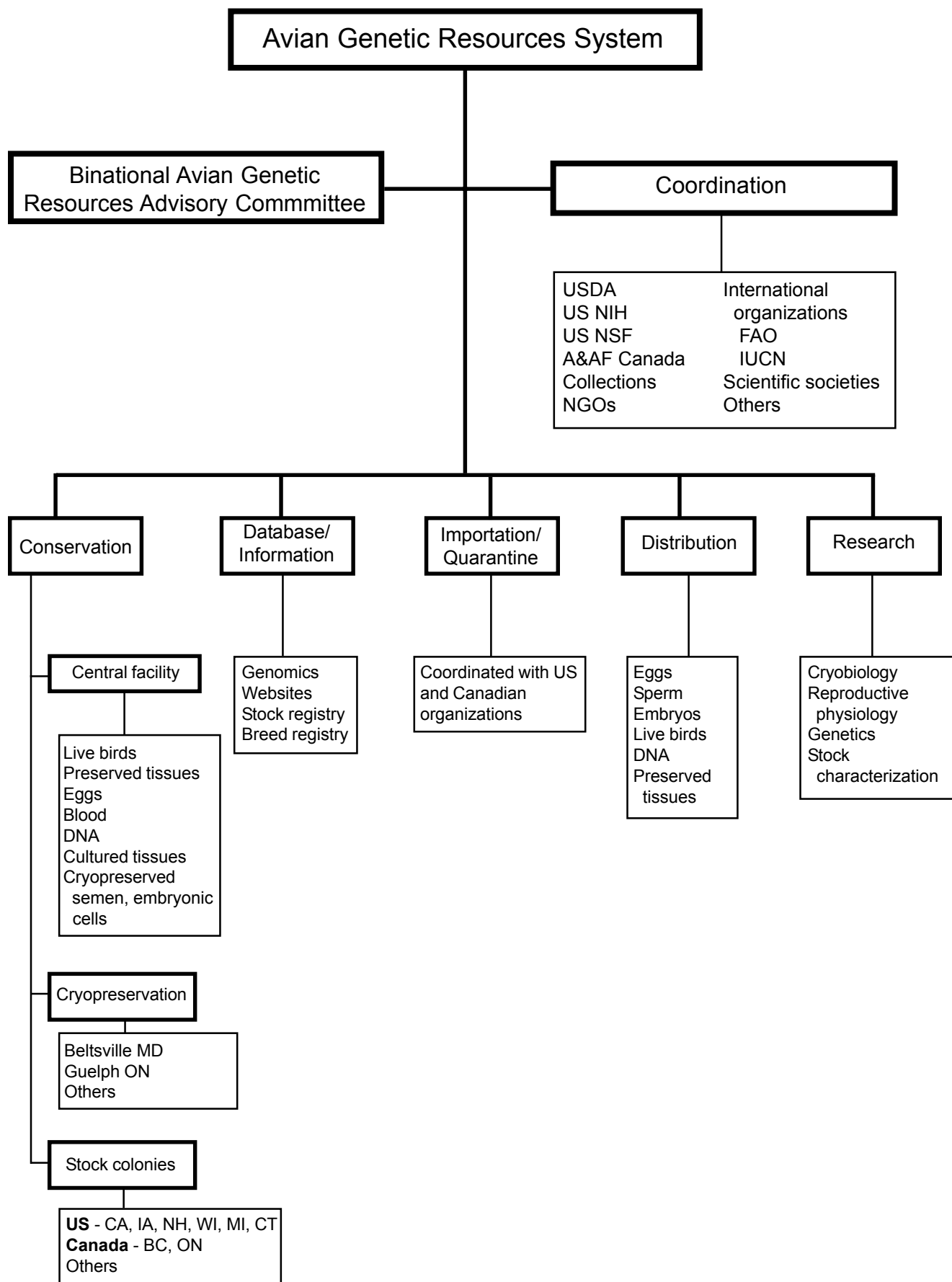
Components of an Avian Genetic Resources System

The Avian Genetic Resources System is envisioned as a multilocal organization that would serve the avian genetic resources needs for the US and Canada. The AVGRS would feature a central facility as a focal point for many of the activities of the System. The functional components are outlined in Figure 16 and are briefly discussed here.

Avian Genetic Resources Advisory Committee (AVGRAC)

The AVGRS would be advised by this binational committee comprised of representatives of national and state/provincial agencies, stock curators, and researchers. It would consist of 12 to 15 individuals who have worked with avian genetic resource issues, drawn from government, industry, and academic institutions in the US and Canada. Specifically, they should have worked in close association with national, international and private research-oriented organizations, and be familiar with avian genetic stock conservation issues. The members should meet at least once a year and be in regular communication during the year. The Committee would review reports and recommendations for conservation of stocks received from species-oriented committees. It would make recommendations to the management unit of the AVGRS.

Figure 16. Components of the Avian Genetic Resources System.



Coordination

The various government and research institutions involved in avian research and conservation would use the AVGRS for coordination of information about genetic resources and the AVGRS would in turn be responsible for maintaining and distributing this information. This function would also include strategic planning for conservation of particular stocks, based on advice from advisory groups established for each species. For example, imperiled stocks would be identified to the AVGRS and a plan for their conservation would be developed through coordinated analysis. International relationships would be coordinated through the AVGRS, including conservation of stocks in other countries, import and export of genetic stocks, data sharing, and development of conservation plans for landraces, wild species, and breeding populations.

Conservation

This is the cornerstone activity of the AVGRS and of critical importance. A central facility is needed for conservation and distribution of genetic materials. The central facility would house those living genetic stocks that could not be maintained elsewhere and would serve as a backup site for important stocks that are maintained elsewhere. This would include a secure backup repository for privately owned lines or populations, either as live birds or cryopreserved germplasm at the central or secondary centers on a fee basis. The central facility would also physically maintain the various types of preserved genetic resources and would coordinate those maintained elsewhere. The cryopreservation capabilities of the central facility would be supplemented by a specialized cryopreservation center, presently unused, at the USDA site in Beltsville, Maryland. No site for the central facility is identified at this time.

The central facility would support and be linked to secondary facilities located at active research centers which have the capability of maintaining genetic stocks for their own research needs. Several locations in the US and Canada would qualify as secondary sites.

Methods of conservation. Conservation methods employed in the AVGRS would be live-bird maintenance and cryopreservation.

Targets for conservation. Conservation emphasis of the AVGRS should be on live birds, embryonic cells, gametes, DNA, and tissues. The target species for the system will be those of interest in

agriculture for food production and for basic biological and biomedical research. Thus, the focus will be on chicken, turkey, and Japanese quail genetic stocks. However, this system could also consider wild turkey, jungle fowl, and game birds, as well as other species commonly raised in captivity. The AVGRS will emphasize:

- Genetic stocks having traits and genetic characteristics useful in research, such as inbred lines, single-gene mutations, chromosome aneuploidy, and DNA marker sequences.
- Lines and populations developed by private and public breeders by hybridization and selection for important production-related traits.
- Domesticated mid-level production and fancy breeds held by small producers and hobbyists in North America and Europe.
- Domesticated, but primitive, landraces existing in Asia, Central and South America, and elsewhere, primarily as scavenger birds.

Archival preserved specimens of birds, organs, skeletons, eggs, feathers, and tissues that have been preserved as museum specimens are also a component of the genetic resources system, since these materials provide for baseline observations and time-course monitoring of factors such as environmental toxicants.

An informal *in situ* system of conservation of landraces and breeds is well established in North America. A monitoring and database system may be the most important need for those genetic resources. This could become an activity of this proposed system.

Databases

Detailed information about all genetic stocks in the US and Canada should be maintained and updated by the AVGRS in a genetic resources information system, similar to the Genetic Resources Information Network (GRIN) developed for the US National Plant Germplasm System and housed with the USDA National Agriculture Library. For example, all of the information included in Appendix 2 should become part of the AVGRS database. Additionally, database service would be offered to the various breed conservancies and hobbyists groups for inventory and location of conserved breeds, land-races, and spe-

cialty stocks. It would also be logical for the AVGRS database to include DNA sequence data as they are developed.

While GRIN currently focuses on plant information, its goal is to include information on all of the common and endangered breeds of farm animals, including the avian genetic stocks used primarily in research. Collaboration with the AVGRS database would facilitate this goal.

Outreach

Researchers can also be informed of the wide variety of available genetic stocks at the annual meetings of a variety of organizations, including the Poultry Science Association, the Pacific Egg and Poultry Association, the Poultry Breeders Roundtable, the Society for Developmental Biology, the American Association for the Advancement of Science, and the American Medical Association. Presentations at the commercially oriented meetings could be used to showcase the benefits the different companies could derive from supporting an avian genetic stocks conservation program, while the basic research or disease model aspect of genetic stocks could be emphasized for organizations promoting basic and biomedical research. Thus, the underlying goals of presenting genetic stocks information at such meetings is not only to attract the attention of researchers, but to engage the interest and promote funding from commercial sectors that can benefit directly from research using avian genetic stocks.

Another outreach option for the AVGRS would be an independent website that would promote the available avian genetic stocks to the scientific community by advertising what is available, and indicating those that are slated for elimination. The effectiveness of such a site could be multiplied by linking it with websites: the animal genetic map (Angen), the chicken map (ChickMap), various research organization sites (e.g., Poultry Science Association, Society for Developmental Biology, various commercial poultry sites, and sites for academic institutions).

The AVGRS website information could be further promoted by a series of clearly written review articles in several of the major biological science journals. In each case, a specific area of research would be targeted, such as animal models for human diseases, limb pattern defects, craniofacial defects, integumentary defects, or immunogenetic research.

The outreach activity would also involve international contacts through FAO or various countries with respect to avian genetic resources. For example, there are genetic stocks at risk in other

countries that should be considered for rescue in the AVGRS because of their value for research.

Bird care and housing

The housing and care of the live birds at all centers will follow American Association for Laboratory Animal Care (AALAC) standards for a breeding colony. Essentially, the breeding stock should be kept in a facility that approximates that of a well-run commercial poultry breeding farm. The highest degree of automation for feeding, cleaning, watering and climate control is recommended. With most of the chicken genetic stocks, the adult birds will be housed in single-bird cages, and bred by artificial insemination. Other features of the facility may include: floor pens, with or without trap nests, to maintain stocks that do not perform well in cages; and separation of males and the females (in different rooms or cage rows), so that appropriate, sex-specific breeder diets, lighting, and feeding regimens can be provided for each group.

To minimize the disease problems in the genetic stocks and reduce the risk of spreading pathogens when stocks are shipped, all conservation centers should follow the National Poultry Improvement Plan guidelines (USDA-APHIS 1998). These procedures impact on the design of the facility, such as:

- All incoming stocks should be acquired only as fertile eggs, which are formaldehyde-fumigated, then incubated, hatched, and reared in a facility isolated from all other avian species, including those already shown to be free of the targeted disease agents. After the birds in isolation are at least six weeks old, they should be blood-tested for the presence of egg-transmitted pathogens (in chickens, this usually includes *Salmonella pullorum*, *S. typhimurium*, *Mycoplasma gallisepticum*, and *M. synoviae*; turkeys would also be tested for *M. gallinarium*), before being moved to the stock center.
- A random-sample of 10% of the adult birds should be tested each year for evidence of the important poultry diseases, including the disease pathogens listed above.
- Replacement birds should be raised in strict isolation for the first month after hatch.
- The stock center should be physically well-isolated from other poultry (commer-

cial or hobby flocks) and from facilities in which other captive bird species are housed.

- The bird houses should prevent entry of wild birds and vermin.
- Access to the facility should be restricted and closely monitored, complying with full necessary sanitary restrictions.

Importation and quarantine

Movement of animals introduces risks of spreading contagious diseases, obviously of great concern in long-term conservation of live birds. Movement of genetic resources as fertile eggs or semen reduces disease transmission risk and are preferred procedures. Importation of stocks to the central facility will be done through on-site isolation and through national facilities under the direction of USDA Animal and Plant Health Inspection Service. The central facility will have appropriate isolation and sanitation capabilities.

Distribution

A major function of the AVGRS will be to provide genetic materials to users in the research community, breeders, and others. The genetic stocks may be transferred as live birds, semen, or eggs. These will be distributed on a cost-recovery basis. Some users require a continuing supply of genetic stocks and these needs would be supplied by contractual stock reproduction programs. The distribution function would supply well-documented stocks to researchers, thus contributing to the integrity of research projects. This functional component of the AVGRS is analogous to services provided by the Jackson Laboratories for mouse genetic stocks.

Research

The AVGRS should have a research capability within the central facility, especially for developing cryopreservation technologies. Research would also be done on methods for documenting genetic integrity or diagnostics with DNA markers. Other research would be done as needed and appropriate. The research activities would be networked with research laboratories in the US and Canada for collaborative work.

Facilities and organizational aspects of AVGRS

The central facility

Ideally, the primary AVGRS facility would be constructed *de novo* near or part of a major agricultural institution (land-grant university) with a veterinary school that has a good avian medicine program, but reasonably isolated from commercial poultry stocks. The connection with a land-grant institution would give the center close ties to active research laboratories and faculty, who could benefit from such a resource and be drawn upon in support of the center. Access to state-of-the-art poultry disease diagnostics and veterinary care is critical, along with good, off-site quarantine facilities for newly acquired genetic stocks. Locating in a strong poultry producing state would also provide an existing poultry-oriented political and commercial infrastructure that could be mobilized to help support the conservation center.

This facility would include a hatchery, brooding and growing areas, adult bird housing, an isolation area, a cryopreservation laboratory and storage facility, a database center, staff and administrative offices, and a laboratory to support research and analytical services.

Bird housing should be designed to minimize disease and pest control problems, and maximize caretaker efficiency and bird well-being. Initially the facility should accommodate 1,000 adult birds, with options for expansion. Several design schemes will be evaluated to accommodate the genetic diversity of the stocks and their special requirements and for efficiency in management.

Network of secondary genetic stock centers

Secondary stock centers would be designated as part of the AVGRS at land-grant universities and other institutions across the US and Canada that fulfilled two criteria: (1) had adequate facilities and support for the genetic stocks used in its own research programs and (2) had a long-term interest in conserving genetic stocks. Such centers, approved as part of the AVGRS, would receive funding from AVGRS for maintenance of stocks. These secondary centers would maintain live birds and would provide backup for at-risk stocks held in the central facility. As with the central facility, the secondary stock colonies would also have a distribution function on a fee basis. Researchers could also, on a fee basis,

arrange to keep research flocks at the central facility or one of the secondary centers, since they may find it difficult or impossible to keep such stocks at their own institutions.

Secondary centers could specialize on one or a few classes of genetic stocks (e.g., inbred strains, congenic series, randombred stocks, selected stocks, single-gene mutant stocks, or chromosome translocation stocks). In fact, many of the existing poultry collections at both academic and government institutions are already quite specialized. The restricted number of classes and species of stocks at each secondary live bird center would simplify reproduction and maintenance for the curators, since each type of stock often has different needs in relation to population sizes, selection criteria, inbreeding considerations, and testing techniques.

Management

The central facility and the secondary centers would be administered by the research institutions with which they are located. The central facility would have, at a minimum, a director or manager (research scientist), curator, an administrative assistant, database manager, cryopreservation research scientist, and three or four laboratory and animal care technicians.

The AVGRS would be guided by the advisory committee on such matters as:

- Information system and website management
- Inventory control
- Conservation status determination (cryopreserved stocks only)
- Stock accessioning strategies
- Importation targets and necessary international cooperation agreements
- Appropriate stock-specific conservation protocols
- Management of grant funds for research and preservation efforts
- Curator training programs
- Program expansion decisions
- Budgets
- Fund raising

- Species coordinating committee organization
- Promotion of the use of these genetic stocks
- User fees and maintenance contract guidelines

Stock evaluation guidelines

One of the more important activities of the AVGRAC would be the evaluation of stocks for conservation, cryopreservation, or elimination. Guidelines for assessing the value of genetic stocks have been outlined in a report by the National Research Council Committee on Preservation of Laboratory Animal Resources (NRC 1990). The report suggests that value of a given stock should be established by considering:

1. Importance of the disease process or physiological function exemplified by the stock (especially when used as an animal model for a human disorder).
2. Validity of the model and continued genetic integrity of the stock.
3. Replaceability of the stocks (those developed after many years of selection or arising from a spontaneous mutation would be considered relatively irreplaceable).
4. Versatility of the stock (the variety of problems that can be addressed with a given stock).
5. Number of users.

The advisory committee would also formulate other criteria specifically relevant for the conservation of avian species.

Financing the Avian Genetic Resources System

Multiple sources of funding will be necessary to meet all of the needs of the AVGRS. Initial costs are those to construct the central facility and upgrade the secondary stock centers. Annual costs of the central facility would be for its personnel and operations. It would also be necessary to support the annual activity of the AVGRAC. The central facility could also direct

funds for specific needs to the secondary centers by means of annual grants.

From the US side, the biological resource programs of the National Science Foundation and the National Institutes of Health would be expected to provide operational funds through direct grants and through grants to investigators who use the avian genetic resources in their research. The USDA's National Genetic Resources System should participate in the AVGRS through the Agricultural Research Service and the Cooperative State Research, Extension, and Education Service. The various State Agricultural Experiment Stations and land-grant and other Universities should also participate. Canadian support and participation should be forthcoming to the extent that the AVGRS provides support to its research and development programs.

The AVGRS will be the major provider of genetic materials to researchers throughout the public and private sectors. For the most part, these researchers do not have capacity to maintain live bird colonies and depend upon stock colonies for their research. User-fees are an appropriate means to recoup costs of stock maintenance.

Donation funds can be expected to support the perpetual maintenance of particular genetic stocks. These funds may be provided as annual grants or through income derived from interest on endowment accounts.

Funding should be sought from the US government for construction of the central facility and for personnel support for operations as a part of the US National Animal Genetic Resources System.

Long-term funding would be the most secure from endowment funds. Contributors could be encouraged from the private sector, from large integrated commercial poultry companies to private individuals with interests in preserving poultry stocks or willing to promote the conservation of stocks that can be used to study human diseases.

Construction, personnel, and operational costs have not been established, pending further analysis of potential sites for the central facility and other considerations. For illustrative purposes, rough order-of-magnitude estimates are given in Table 4.

Table 4. Estimated costs of Avian Genetic Resources System.

Startup costs	
Constructing and equipping central facility	\$15,000,000
Upgrading and renovating secondary centers	2,000,000
Total	\$17,000,000
Annual costs	
Personnel at central facility	\$400,000
Operating costs at central facility	100,000
Grants to secondary centers (8 x \$25,000)	200,000
Advisory Committee	25,000
Total	\$725,000

References

- AA-FC. 1996. Poultry Market Review-1996. Agriculture and Agri-Food Canada. <http://www.agr.ca/misb/aisd/poultry/pa96come.htm>.
- AARTS, H.J., M.C. VAN DER HULST-VAN ARKEL, G. BEUVING, and F.R. LEENSTRA. 1991. Variations in endogenous viral gene patterns in White Leghorn, medium heavy, White Plymouth Rock, and Cornish chickens. *Poult. Sci.* **70**:1281-1286.
- ABBOTT, U.K. 1967. Avian developmental genetics. p. 12-52 *In*: F.W. Will and N.K. Wessels (eds.) *Methods in Developmental Biology*. Thomas Y. Crowell Co. New York.
- ABBOTT, U.K. and V.S. ASMUNDSON. 1957. Scaleless, an inherited ectodermal defect in domestic fowl. *J. Hered.* **48**:63-70.
- ABPLANALP, H. 1992. Inbred lines as genetic resources of chickens. *Poult. Sci. Rev.* **4**:29-39.
- ABPLANALP, H., M.E. GERSHWIN, E. JOHNSTON, and J. REID. 1990. Genetic control of avian scleroderma. *Immunogenetics* **31**:291-295.
- ALDERSON, L. (ed.) 1990. *Genetic Conservation of Domestic Livestock, V. 1*. C.A.B. International. Oxon, UK.
- ANDERSSON, L., A. ARCHIBALD, M. ASHBURNER, S. AUDUN, W. BARENDSE, J. BITGOOD, C. BOTTEMA, T. BROAD, S. BROWN, D. BURT, C. CHARLIER, N. COPELAND, S. DAVIS, M. DAVISSON, J. EDWARDS, A. EGGEN, G. ELGAR, J.T. EPPIG, I. FRANKLIN, P. GREVE, T. GILL III, J.A.M. GRAVES, R. HAWKEN, J. HETZEL, A. HILYARD, H. JACOB, L. JASWINSKA, N. JENKINS, H. KUNZ, G. LEVAN, O. LIE, L. LYONS, P. MACCARONE, C. MELLERSH, G. MONTGOMERY, S. MOORE, C. MORAN, D. MORIZOT, M. NEFF, F. NICHOLAS, S. O'BRIEN, Y. PARSONS, J. PETERS, J. POSTLETHWAIT, M. RAYMOND, M. ROTHSCHILD, S. SCHOOK, Y. SUGIMOTO, C. SZPIRER, M. TATE, J. TAYLOR, J. VANDEBERG, M. WAKEFIELD, J. WIENBERG, and J. WOMACK. 1996. Comparative genome organization of vertebrates. The First International Workshop on Comparative Genome Organization. *Mammalian Genome* **7**:717-734.
- APA. 1998. *The American Standard of Perfection*. American Poultry Association, Inc. Mendon, MA.
- ARS. 1960. *Random sample egg production tests, 1958-1959 combined summary*. ARS 44-79. USDA Agricultural Research Service. Washington, DC.
- ARTHUR, J.A. 1986. An evaluation of industry breeding programs for egg type chickens. p. 325-336 *In*: *Third World Congress on Genetics Applied to Livestock Production. X. Breeding Programs for Swine, Poultry, and Fish*. July 16-22, 1986. Lincoln, NE.
- ASHMORE C.R. and R.G. SOMES JR. 1966. Delay of hereditary muscular dystrophy of the chicken by oxygen therapy. *Proc. Soc. Exp. Biol. Med.* **122**:1100-1103.
- AUSTIN, L.M., R.E. BOISSY, B.S. JACOBSON, and J.R. SMYTH JR. 1992. The detection of melanocyte autoantibodies in the Smyth chicken model for vitiligo. *Clin. Immunol. Immunopath.* **64**:112-120.
- AUSTIN, L.M. and R.E. BOISSY. 1995. Mammalian tyrosinase-related protein-1 is recognized by autoantibodies from vitiliginous Smyth chickens - an avian model for human vitiligo. *Am. J. Pathol.* **146**:1529-1541.
- BACON, L.D. 1987. Influence of the major histocompatibility complex on disease resistance and productivity. *Poult. Sci.* **66**:802-811.
- BACON, L.D. and R.R. DIETERT. 1991. Genetic control of cell-mediated immunity in chickens. *Poult. Sci.* **70**:1187-1199.
- BACON, L.D. and R.L. WITTER. 1992. Influence of turkey herpesvirus vaccination on the B-haplotype: effect on Marek's disease resistance in 15.B-congenic chickens. *Avian Dis.* **36**:378-385.

- BACON, L.D. and R.L. WITTER. 1995. Efficacy of Marek's disease vaccines in MHC heterozygous chickens- MHC congenic X inbred line F-1 matings. *J. Hered.* **86**:269-273.
- BAKST, M.R. 1990. Preservation of avian cells. p. 91-108 *In*: R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- BAKST, M.R. 1993. The anatomy of reproduction in birds, with emphasis on poultry. p. 15-28 *In*: R.J. Etches and A.M. Verrinder Gibbins (eds.) *Manipulation of the Avian Genome*. CRC Press. Boca Raton, FL.
- BATESON, W. 1902. Experiments with poultry. *Poultry Repts. Evol. Comm. Royal Soc.* **1**:87-124.
- BATESON, W. and R.C. PUNNETT. 1906. Experimental studies in the physiology of heredity. *Poultry Repts. Evol. Comm. Royal Soc.* **3**:11-30.
- BATESON, W. and R.C. PUNNETT. 1908. Experimental studies in the physiology of heredity. *Poultry Repts. Evol. Comm. Royal Soc.* **4**:18-35.
- BIXBY, D.E., C.J. CHRISTMAN, C.J. EHRLMAN, and D.P. SPONENBERG. 1994. *Taking Stock: The North American Livestock Census*. The McDonald & Woodward Publishing Co. Blacksburg, VA.
- BLOOM, S.E. and L.D. BACON. 1985. Linkage of the major histocompatibility (B) complex and the nucleolar organizer in the chicken. Assignment to a microchromosome. *J. Hered.* **76**:146-154.
- BOWERS, R.R., J. LUJAN, A. BIBOSO, S. KRIDEL, and C. VARKEY. 1994. Premature avian melanocyte death due to low antioxidant levels of protection: Fowl model for vitiligo. *Pigment Cell Res.* **7**:409-418.
- BRILES, W.E., R.M. GOTO, C. AUFRAY, and M.M. MILLER. 1993. A polymorphic system related to but genetically independent of the chicken major histocompatibility complex. *Immunogenetics* **37**:408-414.
- BROTMAN, H.F. 1977a. Abnormal morphogenesis of feather structures and pattern in the chick embryo integument. II. Histological description. *J. Exp. Zool.* **200**:107-124.
- BROTMAN, H.F. 1977b. Epidermal-dermal tissue interactions between mutant and normal embryonic back skin: site of mutant gene activity determining abnormal feathering is the epidermis. *J. Exp. Zool.* **200**:125-136.
- BUMSTEAD, N. and J. PALYGA. 1992. A preliminary linkage map of the chicken genome. *Genomics* **13**:690-697.
- BURT, D.W., N. BUMSTEAD, J.J. BITGOOD, F.A. PONCE DE LEON, and L.B. CRITTENDEN. 1995. Chicken genome mapping: a new era in avian genetics. *Trends Genet.* **11**:190-194.
- BUSS, E.G. 1993. Cryopreservation of rooster semen. *Poult. Sci.* **72**:944-954.
- CARSIENCE, R.S., M.E. CLARK, A.M. VERRINDER GIBBINS, and R.J. ETCHES. 1993. Germline chimeric chickens from dispersed donor blastodermal cells and compromised recipient embryos. *Development* **117**:669-675.
- CAST. 1984. Animal germplasm preservation and utilization in agriculture. Report No. 101. Council for Agricultural Science and Technology. Ames, IA.
- CHANG, I.K., A. YOSHIKI, M. KUSAKABE, A. TAJIMA, T. CHIKAMUNE, M. NAITO, and T. OHNO. 1995. Germ line chimera produced by transfer of cultured chick primordial germ cells. *Cell Biol. Internat.* **19**:569-576.
- CHENG, H.H. 1997. Mapping the chicken genome. *Poult. Sci.* **76**:1101-1107.
- CHENG, H.H., I. LEVIN, R.L. VALLEJO, H. KHATIB, J.B. DODGSON, L.B. CRITTENDEN, and J. HILLEL. 1995. Development of a genetic map of the chicken with markers of high utility. *Poult. Sci.* **74**:1855-1874.
- CHENG, K.M. and M. KIMURA. 1990. Mutations and major variants in Japanese quail. p. 333-362 *In*: R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- CHUONG, C.-M. (ed.) 1998. *Molecular Basis of Epithelial Appendage Morphogenesis*. R.G. Landes Co. Austin, TX.
- CONNELLY, T.G., L.L. BRINKLEY, and B.M. CARLSON (eds.) 1981. *Morphogenesis and Pattern Formation*. Raven Press. New York.
- COPELAND, N.G. and N.A. JENKINS. 1991. Development and applications of a molecular genetic linkage map of the mouse genome. *Trends Genet.* **7**:113-118.
- CRAWFORD, R.D. 1990. Poultry genetic resources: evolution, diversity, and conservation. p. 43-60 *In*: R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- CRAWFORD R.D. and C. CHRISTMAN. 1992. Heritage hatchery networks in poultry conservation. p. 212-222 *In*: L. Alderson and I. Bodo (eds.) *Genetic Conservation of Domestic Livestock*, V. 2. C.A.B. International. Oxon, UK.
- CRITTENDEN, L.B., W. OKAZAKI and R. REAMER. 1963. Genetic resistance to Rous sarcoma virus in embryo cell cultures and embryos. *Virology* **20**:541-544.
- CRITTENDEN, L.B., L. PROVENCHER, L. SANTANGELO, I. LEVIN, H. ABPLANALP, R.W. BRILES, W.E. BRILES, and J.B. DODGSON. 1993. Characterization of a Red Jungle Fowl by White Leghorn backcross reference population for mo-

- lecular mapping of the chicken genome. *Poult. Sci.* **72**:334–348.
- CROOIJMANS, R.P.M.A., P.A.M. VAN OERS, J.A. STRIJK, J.J. VAN DER POEL, and M.A.M. GROENEN. 1996. Preliminary linkage map of the chicken (*Gallus domesticus*) genome based on microsatellite markers: 77 new markers mapped. *Poult. Sci.* **75**:746–754.
- CUNHA, G.R. 1994. Role of mesenchymal-epithelial interactions in normal and abnormal development of the mammary gland and prostate. *Cancer* **74**:1030–1044.
- DELANY, M.E., R.R. DIETERT, and S.E. BLOOM. 1988. MHC-chromosome dosage effects: evidence for increased expression and alteration of B cell subpopulations in neonatal aneuploid chickens. *Immunogenetics* **27**:24–30.
- DELANY, M.E., D.E. MUSCARELLA, and S.E. BLOOM. 1994. Effects of rRNA gene copy number and nucleolar variation on early development: inhibition of gastrulation in rDNA-deficient chick embryos. *J. Hered.* **85**:211–217.
- DELANY, M.E. and J.M. PISENTI. 1998. Conservation of poultry genetic research resources: Consideration of the past, present and future. *Poult. and Avian Biol. Rev.* **9**:25–42.
- DELANY, M.E., R.L. TAYLOR Jr., and S.E. BLOOM. 1995. Teratogenic development in chicken embryos associated with a major deletion in the rRNA gene cluster. *Dev. Growth Differ.* **37**:403–412.
- DIETERLEN-LIEVRE, F. 1997. Avian models in developmental biology. *Poult. Sci.* **76**:78–82.
- DIETERLEN-LIEVRE F. and N. LEDOUARIN. 1993. The use of avian chimeras in developmental biology. p. 103–120 *In*: R.J. Etches and A.M. Verrinder Gibbins (eds.) *Manipulation of the Avian Genome*. CRC Press. Boca Raton, FL.
- DODGSON, J.B., J. STROMMER and J. D. ENGEL. 1979. The isolation of the chicken β -globin gene and a linked embryonic β -like globin gene from a chicken DNA recombinant library. *Cell* **17**:879–887.
- DUNNINGTON, E.A., L.C. STALLARD, J. HILLEL, and P.B. SIEGEL. 1994. Genetic diversity among commercial chicken populations estimated from DNA fingerprints. *Poult. Sci.* **73**:1218–1225.
- EDE, D.A., J.R. HINCHLIFFE, and M. BALLS (eds.) 1977. *Vertebrate Limb and Somite Morphogenesis*. Cambridge University Press. Cambridge, UK.
- EMSLEY, A. 1993. Aspirations of breeders in the poultry industry as manipulation of the avian genome continues. p. 275–284. *In*: R.J. Etches and A.M. Verrinder Gibbins (eds.) *Manipulation of the Avian Genome*. CRC Press. Boca Raton, FL.
- ENGEL, J. D. and J. B. DODGSON. 1981. Histone genes are clustered but not tandemly repeated in the chicken genome. *Proc. Natl. Acad. Sci. (USA)* **78**:2856–2860.
- ERF, G.F., A.V. TREJO-SKALLI, and J.R. SMYTH JR. 1995. T cells in regenerating feathers of Smyth line chickens with vitiligo. *Clin. Immunol. Immunopathol.* **76**:120–126.
- EYAL-GILADI, H. and S. KOCHAV. 1976. From cleavage to primitive streak formation: a complementary normal table and a new look at the first stage of the development of the chick. I. General morphology. *Dev. Biol.* **49**:321–337.
- FALLON, J.F., P.F. GOETINCK, R.O. KELLEY, and D.L. STOCUM (eds.) 1993. *Limb Development and Regeneration*, Pt A&B. Proc. 4th Intl. Conf. on Limb Development and Regeneration, Asilomar, CA, July 1992. Wiley-Liss. New York.
- FALLON, J.F., A. LOPEZ, M.A. ROS, M.P. SAVAGE, B.O. OLWIN, and B.K. SIMANDL. 1994. FGF-2: apical ectodermal ridge growth signal for chick limb development. *Science* **264**:104–107.
- FAO. 1992. *Expert consultation on the management of global animal genetic resources*. Food and Agriculture Organization Report, Rome, Italy, 7–10 April, 1992.
- FUMIHITO, A., S. MIYAKE, M. SUMI, M. TAKADA, S. OHNO, and N. KONDO. 1994. One subspecies of the Red Jungle Fowl (*Gallus gallus gallus*) suffices as the matriarchic ancestor of all domestic breeds. *Proc. Natl. Acad. Sci. (USA)* **91**:12505–12509.
- GAVORA, J.S. 1990. Disease genetics. p. 805–846 *In*: R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- GEE, G.F. 1995. Artificial insemination and cryopreservation of semen from nondomestic birds. p. 262–279 *In*: M.R. Bakst and G.J. Wishart (eds.) *Proceedings: First International Symposium on the Artificial Insemination of Poultry*. Poultry Science Association. Illinois.
- GERSHWIN, M.E., H. ABPLANALP, J.J. CASTLES, R.M. IKEDA, J. VAN DER WATER, J. EKLUND, and D. HAYNES. 1981. Characterization of a spontaneous disease of White Leghorn chickens resembling progressive systemic sclerosis (scleroderma). *J. Exp. Med.* **153**:1640–1659.
- GOETINCK, P.F. and U.K. ABBOTT. 1963. Tissue interactions in the scaleless mutant and the use of scaleless as an ectodermal marker in studies of normal limb differentiation. *J. Exp. Zool.* **154**:7–19.
- GOETINCK, P.F. and M.J. SEKELICK. 1972. Observations on collagen synthesis, lattice formation, and morphology of scaleless and normal embryonic skin. *Dev. Biol.* **28**:636–648.

- GRIESHAMMER, U., G. MINOWADA, J.M. PISENTI, U.K. ABBOTT, and G.R. MARTIN. 1996. The chick limbless mutation causes abnormalities in limb bud dorsal-ventral patterning: implications for the mechanism of apical ridge formation. *Development* **121**:3851–3861.
- GRUNDER, A.A., B. BENKEL, J.R. CHAMBERS, M.P. SABOUR, and J.S. GAVORA. 1995. Characterization of eight endogenous viral (*ev*) genes of meat-chickens in semi-congenic lines. *Poult. Sci.* **74**:1506–1514.
- GRUNDER, A.A., B. BENKEL, P. SABOUR, and J.S. GAVORA. 1993. Research Note: Avian leukosis retroviral genes are not detected in geese. *Poult. Sci.* **72**:363–367.
- GRUNDER, A.A. and B. PAWLUCZUK. 1991. Comparison of procedures for collecting semen from ganders and inseminating geese. *Poult. Sci.* **70**:1975–1980.
- GRUNDER, A.A., M.P. SABOUR, and J.S. GAVORA. 1994. Estimates of relatedness and inbreeding in goose strains from DNA fingerprints. *Animal Genet.* **25**(Suppl. 1):81–88.
- GUILLEMOT, F., J.F. KAUFMAN, K. SKJOEDT, and C. AUFRAY. 1989. The major histocompatibility complex in the chicken. *Trends Genet.* **5**:300–304.
- HAMBURGER, V. and H.L. HAMILTON. 1951. A series of normal stages in the development of the chick embryo. *J. Exp. Morphol.* **88**:49–92.
- HAMMERSTEDT, R.H. 1995. Cryopreservation of poultry semen - current status and economics. p. 229–250 *In*: M.R. Bakst and G.J. Wishart (eds.) *Proceedings: First International Symposium on the Artificial Insemination of Poultry*. Poultry Science Association. Illinois.
- HAMMERSTEDT, R.H. and J.K. GRAHAM. 1992. Cryopreservation of poultry sperm: the enigma of glycerol. *Cryobiol.* **29**:26–28.
- HARPER, J.A., P.E. BERNIER, and L.L. THOMPSON-COWLEY. 1983. Early expression of hereditary deep pectoral myopathy in turkeys due to forced wing exercise. *Poult. Sci.* **62**:2303–2308.
- HAWES, R.O. 1990. Mutants and major variants in geese. p. 395–400 *In*: R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- HELLER, E.D., Z. UNI, and L.D. BACON. 1991. Serological evidence for major histocompatibility complex (B-complex) antigens in broilers selected for humoral immune response. *Poult. Sci.* **70**:726–732.
- HEMENDINGER R.A., J.R. PUTNAM, and S.E. BLOOM. 1992. MHC dosage effects on primary immune organ development in the chicken. *Devel. Compar. Immun.* **16**:175–186.
- HEMENDINGER, R.A., M.M. MILLER, and S.E. BLOOM. 1995. Selective expression of major histocompatibility complex (MHC) antigens and modulation of T-cell differentiation in chickens with increased MHC-chromosome dosages. *Vet. Immunol. Pathol.* **46**:303–316.
- HINCHLIFFE, J.R. and D.R. JOHNSON. 1980. *The Development of the Vertebrate Limb: An approach through experiment, genetics, and evolution*. Clarendon Press. Oxford, UK.
- HUNTON, P. 1990. Industrial breeding and selection. p. 985–1028 *In*: R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- HUTT, F.B. 1936. Genetics of the fowl. VI. A tentative chromosome map. p. 105–112 *In*: *Neue Forschungen Tierzucht und Abstammungslehre* (Duerst Festschrift).
- HUTT, F.B. 1949. *Genetics of the Fowl*. McGraw-Hill Book Co. New York.
- IANNACCONE, S., A. QUATTRINI, S. SMIRNE, M. SESSA, F. DE RINO, L. FERINI-STRAMBI, and R. NEMNI. 1995. Connective tissue proliferation and growth factors in animal models of Duchenne muscular dystrophy. *J. Neurol. Sci.* **128**:36–44.
- IRAQI, F., M. SOLLER, and J.S. BECKMANN. 1991. Distribution of endogenous viruses in some commercial chicken layer populations. *Poult. Sci.* **70**:665–679.
- JARVI, S.I., R.M. GOTO, W.E. BRILES, AND M.M. MILLER. 1996. Characterization of MHC genes in a multigenerational family of ring-necked pheasants. *Immunogenetics* **43**:125–135.
- KAGAMI, H., M.E. CLARK, A.M. VERRINDER GIBBINS, and R.J. ETCHES. 1995. Sexual differentiation of chimeric chickens containing ZZ and ZW cells in the germline. *Mol. Reprod. Dev.* **42**:379–388.
- KEAN, R.P., W.E. BRILES, A. CAHANER, A.E. FREEMAN, and S.J. LAMONT. 1994. Differences in major histocompatibility complex frequencies after multitrait, divergent selection for immunocompetence. *Poult. Sci.* **73**:7–17.
- KINO, K., B. PAIN, S.P. LEIBO, M. COCHRAN, and R.J. ETCHES. 1997. Production of chicken chimeras from injection of frozen-thawed blastodermal cells. *Poult. Sci.* **76**:753–760.
- KUWANA, T., K. HASIMOTO, A. NAKAMISHI, Y. YASUDA, A. TAJIMA, and M. NAITO. 1996. Long-term culture of avian embryonic cells in vitro. *Int. J. Develop. Biol.* **40**:1061–1064.
- LAKE, P.E. 1986. The history and future of the cryopreservation of avian germplasm. *Poult. Sci.* **65**:1–15.
- LAMONT, S.J. 1994. Poultry immunogenetics: which way do we go? *Poult. Sci.* **73**:1044–1048.
- LANCASTER, F.M. 1990. Mutations and major variants in domestic ducks. p. 381–388 *In*:

- R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- LANDAUER, W. 1967. *The Hatchability of Chicken Eggs as Influenced by Environment and Heredity*. Storrs Agr. Exp. Sta., Monogr. 1.
- LANDAUER, W. 1973. *The Hatchability of Chicken Eggs as Influenced by Environment and Heredity*. Storrs Agr. Exp. Sta., Monogr. 1 Suppl.
- LAUFER, E., R. DAHN, O.E. OROZCO, C.-Y. YEO, J. PISENTI, D. HENRIQUES, U.K. ABBOTT, J.F. FALLON, and C. TABIN. 1997. Expression of *Radical fringe* on limb-bud ectoderm regulates apical ectodermal ridge formation. *Nature* **386**:366–373.
- LEPAGE, K.T., S.E. BLOOM, and R.L. TAYLOR. 1996. Antibody response to sheep red blood cells in a major histocompatibility (B) complex aneuploid line of chickens. *Poult. Sci.* **75**:346–350.
- LERNER, I.M. 1950. *Population Genetics and Animal Improvement*. Cambridge University Press. Cambridge, UK.
- LERNER, I.M. 1958. *The Genetic Basis of Selection*. Wiley & Sons, Inc., New York.
- LEVIN, I., L. SANTANGELO, H. CHENG, L.B. CRITTENDEN, and J.B. DODGSON. 1994. An autosomal genetic linkage map of the chicken. *J. Hered.* **85**:79–85.
- LIEN, Y.H., J. FU, R.B. RUCKER, M. SCHECK, U. ABBOTT, and R. STERN. 1990. Collagen, proteoglycan and hyaluronidase activity in cultures from normal and scoliotic chicken fibroblasts. *Biochim. Biophys. Acta* **1034**:318–325.
- LIN, M., M.H. THORNE, I.C.A. MARTIN, and B.L. SHELDON. 1986. Histology of the gonads of triploid fowls. *Proc. Aust. Soc. Reprod. Biol.* **18**:77.
- LOVE, J.C., GRIBBIN, C. MATHER, and H. SANG. 1994. Transgenic birds by DNA microinjection. *Bio-Technology* **12**:60–63.
- MACCABE, J.A. and J.K. NOVEROSKE. 1997. The control of cell death in the early chick embryo wing bud. *Poult. Sci.* **76**:105–110.
- MASON, I.L. and R.D. CRAWFORD. 1993. Appendix B: Global status of livestock and poultry species. p. 141–169 In: NRC Committee on Managing Global Genetic Resources: Agricultural Imperatives. *Managing Global Genetic Resources: Livestock*. National Academy Press. Washington, DC.
- MERRITT, E.S. 1962. Selection for egg production in geese. p. 83–87 In: *Proc. 12th World's Poultry Congress*, Sydney, Australia.
- MILLER, M.M., R. GOTO, A. BERNOT, R. ZOOROB, C. AUFRAY, N. BUMSTEAD, and W.E. BRILES. 1994. Two Mhc class I and two Mhc class II genes map to the chicken *Rfp-Y* system outside the B complex. *Proc. Natl. Acad. Sci. (USA)* **91**:4397–4401.
- MILLER, M.M., R.M. GOTO, R.L. TAYLOR JR., R. ZOOROB, C. AUFRAY, R.W. BRILES, W.E. BRILES, and S.E. BLOOM. 1996. Assignment of *Rfp-Y* to the chicken major histocompatibility complex/NOR microchromosome and evidence for high-frequency recombination associated with the nucleolar organizer region. *Proc. Natl. Acad. Sci. (USA)* **93**:3958–3962.
- MOCHIDA, J., D.R. BENSON, U. ABBOTT, and R.B. RUCKER. 1993. Neuromorphometric changes in the ventral spinal roots in a scoliotic animal. *Spine* **18**:350–355.
- MORGAN, B.A. 1997. Hox genes and embryonic development. *Poult. Sci.* **76**:96–104.
- MUSCARELLA, D.E., V.M. VOGT, and S.E. BLOOM. 1987. Characterization of ribosomal RNA synthesis in a gene dosage mutant: the relationship of topoisomerase I and chromatin structure to transcriptional activity. *J. Cell Biol.* **105**:1501–1513.
- NAITO, M., A. TAJIMA, T. TAGAMI, Y. YASUDA, and T. KUWANA. 1994a. Preservation of chick primordial germ cells in liquid nitrogen and subsequent production of viable offspring. *J. Reprod. Fertility* **102**:321–325.
- NAITO, M., A. TAJIMA, Y. YASUDA, and T. KUWANA. 1994b. Production of germline chimeric chickens, with high transmission rate of donor-derived gametes, produced by transfer of primordial germ cells. *Molec. Reprod. Development* **39**:153–161.
- NRC. 1990. Important laboratory animal resources: selection criteria and funding mechanisms for their preservation. Committee on Preservation of Laboratory Animal Resources, Institute of Laboratory Animal Resources, National Research Council. *ILAR News* **32**:A1–A32.
- NRC. 1993. *Managing Global Genetic Resources: Livestock*. National Research Council Committee on Managing Global Genetic Resources: Agricultural Imperatives. National Academy Press. Washington, DC.
- NISWANDER, L., S. JEFFREY, G.R. MARTIN, and C. TICKLE. 1994. A positive feedback loop coordinates growth and patterning in the vertebrate limb. *Nature* **371**:609–612.
- NORAMLY, S., J. PISENTI, U. ABBOTT, and B. MORGAN. 1996. Gene expression in the limbless mutant: Polarized gene expression in the absence of *Shh* and an AER. *Dev. Biol.* **179**:339–346.
- NORTH CAROLINA COOPERATIVE EXTENSION SERVICE. 1996. Final report of the 31st North Carolina

- layer performance and management test. Vol. 31, No. 4. Raleigh NC.
- OKAZAKI, W., H.G. PURCHASE and B.R. BURMESTER. 1970. Protection against Marek's disease by vaccination with a herpesvirus of turkeys. *Avian Dis.* **14**:413-429.
- OLDFIELD, M.L. 1984. *The Value of Conserving Genetic Resources*. US Department of the Interior, National Park Service, Washington, DC.
- OLSEN, M.W. 1965. Twelve year summary of selection for parthenogenesis in Beltsville Small White turkeys. *Brit. Poult. Sci.* **6**:1-6.
- PALMOSKI, M.J. and P.F. GOETINCK. 1970. An analysis of the development of conjunctival papillae and scleral ossicles in the eye of the scaleless mutant. *J. Exp. Zool.* **174**:157-164.
- PARDUE, S.L. 1997. Educational opportunities and challenges in poultry science: Impact of resource allocation and industry needs. *Poult. Sci.* **76**:938-943.
- PARK, JH, E.J. HILL, T.H. CHOU, V. LEQUIRE, R. ROELOFS, and C.R. PARK. 1979. Mechanism of action of penicillamine in the treatment of avian muscular dystrophy. *Ann. NY Acad. Sci.* **317**:356-369.
- PATTERSON, L.T. and G.R. DRESSLER. 1994. The regulation of kidney development: new insights from and old model. *Curr. Opin. Genet. Dev.* **4**:696-702.
- PERLER, F., A. EFSTRATIADIS, P. LOMEDICO, W. GILBERT, R. KOLODNER, and J.B. DODGSON. 1980. The evolution of genes: The chicken preproinsulin gene. *Cell* **20**:555-566.
- PESSAH, I.N. and M.J. SCHIEDT. 1990. Early over-expression of low-affinity [3H]ryanodine receptor sites in heavy sarcoplasmic reticulum fraction from dystrophic chicken pectoralis major. *Biochim. Biophys. Acta* **1023**:98-106.
- PETERS, K., S. WERNER, X. LIAO, S. WERT, J. WHITSETT, and L. WILLIAMS. 1994. Targeted expression of a dominant negative FGF receptor blocks branching morphogenesis and epithelial differentiation of the mouse lung. *EMBO J.* **13**:3296-3301.
- PETITTE, J.N., C.L. BRAZOLOT, M.E. CLARK, G. LIU, A.M. VERRINDER GIBBINS, and R.J. ETCHES. 1993. Accessing the genome of the chicken using germline chimeras. p. 81-101 In: R.J. Etches and A.M. Verrinder Gibbins (eds.) *Manipulation of the Avian Genome*. CRC Press. Boca Raton, FL.
- PETITTE, J.N., M.E. CLARK, G. LIU, A.M. VERRINDER GIBBINS, and R.J. ETCHES. 1990. Production of somatic and germline chimeras in the chicken by transfer of early blastodermal cells. *Development* **108**:185-189.
- POLLOCK, D.L. 1999. A geneticist's perspective from within a broiler primary breeder company. *Poult. Sci.* **78**:414-418.
- PRAHLAD, R., G. SKALA, D. JONES, and W. BRILES. 1979. Limbless: A new genetic mutant in the chick. *J. Exp. Zool.* **209**:427-434.
- QURESHI, M.A., S.E. BLOOM, and R.R. DIETERT. 1989. Effects of increased major histocompatibility complex dosage on chicken monocyte-macrophage function. *Proc. Soc. Exp. Biol. Med.* **190**:195-202.
- QURESHI, M.A. and G.B. HAVENSTEIN. 1994. A comparison of the immune performance of a 1991 commercial broiler with a 1957 randed strain when fed typical 1957 and 1991 broiler diets. *Poult. Sci.* **73**:1805-1812.
- QURESHI, M.A. and R.L. TAYLOR JR. 1993. Analysis of macrophage function in Rous sarcoma-induced tumor regressor and progressor 6.B congenic chickens. *Vet. Immunol. Immunopathol.* **37**:285-294.
- REEDY, S.E., S.P. LEIBO, M.E. CLARK, and R.J. ETCHES. 1995. Beyond freezing semen. p. 251-261 In: M.R. Bakst and G.J. Wishart (eds.) *Proceedings: First International Symposium on the Artificial Insemination of Poultry*. Poultry Science Association. Savoy, IL.
- RIDDLE, R.D., R.L. JOHNSON, E. LAUFER, and C. TABIN. 1993. Sonic hedgehog mediates the polarizing activity of the ZPA. *Cell* **75**:1401-1416.
- RODRIGUEZ, C., R. KOS, D. MACIAS, U.K. ABBOTT, and J.C. IZPISUA-BELMONTE. 1996. *Shh*, *HoxD*, *Bmp-2*, and *Fgf-4* gene expression during development of the polydactylous talpid2, diplopodia1, and diplopodia4 mutant chick limb buds. *Devel. Genet.* **19**:26-32.
- ROMANOFF, A.L. 1960. *The Avian Embryo*. MacMillan Co. New York.
- ROMANOFF, A.L. 1972. *Pathogenesis of the Avian Embryo*. Wiley-Interscience. New York.
- ROSE, N.R. 1994. Avian models of autoimmune disease: lessons from the birds. *Poult. Sci.* **73**:984-990.
- ROUS, P. 1911. A sarcoma of the fowl transmissible by an agent separable from the tumor cells. *J. Exp. Med.* **13**:397-411.
- RUCKER, R.B., W. OPSAHL, U.K. ABBOTT, C. GREVE, C. KENNEY, and R. STERN. 1986. Scoliosis in chickens. A model for the inherited form of adolescent scoliosis. *Am. J. Pathol.* **123**:585-588.
- SALTER, D.W., E.J. SMITH, S.H. HUGHES, S.E. WRIGHT, and L.B. CRITTENDEN. 1987. Trans-genic chickens: insertion of retroviral genes into the chicken germ line. *Virology* **157**:236-240.
- SALTER, D.W., E.J. SMITH, S.H. HUGHES, S.E. WRIGHT, A.M. FADLY, R.L. WITTER, and L.B.

- CRITTENDEN. 1986. Gene insertion into the chicken germ line by retroviruses. *Poult. Sci.* **65**:1445-1458.
- SANDERS, E.J. and M.A. WRIDE. 1997. Roles for growth and differentiation factors in avian embryonic development. *Poult. Sci.* **76**:111-117.
- SAUNDERS, J.W., JR. 1948. The proximodistal sequence of origin of the parts of the chick wing and role of the ectoderm. *J. Exp. Zool.* **108**:363-404.
- SAUNDERS, J.W., JR. 1972. Developmental control of three-dimensional polarity in the avian limb. *Ann. N.Y. Acad. Sci.* **193**:29-42.
- SAWYER, R.H. 1979. Avian scale development: effect of the scaleless gene on morphogenesis and histogenesis. *Dev. Biol.* **68**:1-15.
- SAWYER, R.H. and U.K. ABBOTT. 1972. Defective histogenesis and morphogenesis in the anterior shank skin of the scaleless mutant. *J. Exp. Zool.* **181**:99-110.
- SAWYER, R.H., U.K. ABBOTT, and G.N. FRY. 1974. Avian scale development. IV. Ultrastructure of the anterior shank skin of the scaleless mutant. *J. Exp. Zool.* **190**:71-77.
- SAWYER, R.H. and P.F. GOETINCK. 1988. Developmental genetics of the integument and limb of the domestic chicken. p. 415-439 *In*: G.M. Malacinski (ed.) *Developmental Genetics of Higher Organisms*. Macmillan Publishing Co. New York.
- SCHAT, K.A., R.L. TAYLOR, and W.E. BRILES. 1994. Resistance to Marek's disease in chickens with recombinant haplotypes of the major histocompatibility (B) complex. *Poult. Sci.* **73**:502-508.
- SENGEL, P. 1976. *Morphogenesis of Skin*. Cambridge University Press. Cambridge, UK.
- SENGEL, P. and U.K. ABBOTT. 1963. In vitro studies with scaleless mutant: Interactions during feather and scale differentiation. *J. Hered.* **54**:254-262.
- SEXTON, T.J. 1979. Preservation of poultry semen - a review. p. 159-170 *In*: H.W. Hawk, C.A. Kiddy, and H.C. Cecil (eds.) *Animal Reproduction*. Beltsville Symposium on Agricultural Research, No. 3. John Wiley & Son. New York.
- SGONC, R., H. DIETRICH, M.E. GERSHWIN, A. COLOMBATTI, and G. WICK. 1995. Genomic analysis of collagen and endogenous virus loci in the UCD-200 and 206 lines of chickens, animal models for scleroderma. *J. Autoimmunity* **8**:763-770.
- SILLER, W.G. 1985. Deep pectoral myopathy: a penalty of successful selection for muscle growth. *Poult. Sci.* **64**:1591-1595.
- SOLARI, A.J., M.H. Thorne, B.L. Sheldon, and C.B. Gillies. 1991. Synaptonemal complexes of triploid (ZZW) chickens: Z-Z pairing pre-dominates over Z-W pairing. *Genome* **34**:718-726.
- SOMES, R.G., JR. 1988. *International Registry of Poultry Genetic Stocks*. Storrs Agr. Exp. Sta. Bull. 476. The University of Connecticut.
- SOMES, R.G., JR. 1990. Mutations and major variants in ring-necked pheasants. p. 371-380 *In*: R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- SONG, H.-K. and R.H. SAWYER. 1996. Dorsal dermis of the scaleless (sc/sc) embryo directs normal feather pattern formation until day 8 of development. *Dev. Dynam.* **205**:82-91.
- SPILLMAN, W.J. 1908. Spurious allelomorphism: results of some recent investigations. *Am. Nat.* **42**:610-615.
- STEINER, C., M. MULLER, A. BANIAHMAD and R. RENKAWITZ. 1987. Lysozyme gene activity in chicken macrophages is controlled by positive and negative regulatory elements. *Nucl. Acid Res.* **15**:4163-4178.
- SUNG, A.M., A.W. NORDSKOG, S.J. LAMONT, and C.M. WARNER. 1993. Isolation and characterization of cDNA clones for chicken major histocompatibility complex class II molecules. *Animal Genet.* **24**:227-233.
- TAJIMA, A., M. NAITO, Y. YASUDA, and T. KUWANA. 1993. Production of germ line chimera by transsfer of primordial germ cells in the domestic chicken (*Gallus domesticus*). *Theriogenology* **40**:509-519.
- THORAVAL, P., F. LASSERRE, F. COUDERT, and G. DAMBRINE. 1994. Production of germline chimeras obtained from Brown and White Leghorns by transfer of early blastodermal cells. *Poult. Sci.* **73**:1897-1905.
- THORNE, M.H., R.K. COLLINS, and B.L. SHELDON. 1987. Live haploid-diploid and other unusual mosaic chickens (*Gallus domesticus*). *Cytogenet. Cell Genet.* **45**:21-25.
- THORNE, M.H., R.K. COLLINS, and B.L. SHELDON. 1991. Triploidy and other chromosomal abnormalities in a selected line of chickens. *Genet. Sel. Evol.* **23**(suppl. 1):212s-216s.
- THORNE, M.H., R.K. COLLINS, B.L. SHELDON, and L.W. BOBR. 1988. Morphology of the gonads and reproductive ducts of triploid chickens. *Proc. World Poult. Congr. (Nagoya)* **18**:525-526.
- THORNE, M.H., F.W. NICHOLAS, C. MORAN, and B.L. SHELDON. 1997. Genetic analysis of triploidy in a selected line of chickens. *J. Heredity* **88**:495-498.
- THORNE, M.H. and B.L. SHELDON. 1991. Cytological evidence of maternal meiotic errors in a line of chickens with a high incidence of triploidy. *Cytogenet. Cell Genet.* **57**:206-210.

- THORAVAL, P., M. AFANASSIEFF, F.L. COSSET, F. LASSERRE, G. VERDIER, F. COUDERT, and G. DAMBRINE. 1995. Germline transmission of exogenous genes in chickens using helper-free ectropic avian leukosis virus-based vectors. *Transgenic Res.* **4**:369–377.
- USDA-APHIS. 1997. *National Poultry Improvement Plan and Auxiliary Provisions*. APHIS 91-55-038, USDA Animal and Plant Health Inspection Service. Washington, DC.
- USDA-NASS. 1998. *Agricultural Statistics, 1998*. United States Department of Agriculture, National Agricultural Statistics Service. Government Printing Office, Washington DC.
- VAINIO, S., I. KARAVANOVA, A. JOWETT, and I. THESLEFF. 1993. Identification of BMP-4 as a signal mediating secondary induction between epithelial and mesenchymal tissues during early tooth development. *Cell* **75**:45–58.
- VAN KREY, H.P. 1990. Reproductive biology in relation to breeding and genetics. p. 61–90 *In*: R.D. Crawford (ed.) *Poultry Breeding and Genetics*. Elsevier. New York.
- VAN DE WATER, J., R. BOYD, G. WICK, and M.E. GERSHWIN. 1994. The immunologic and genetic basis of avian scleroderma, an inherited fibrotic disease of line 200 chickens. *Intl. Rev. Immunol.* **11**:273–282.
- VERRINDER GIBBINS, A.M. 1993. Future interactions between molecular biologists and the poultry industry, illustrated by a comparison of the genome mapping and gene targeting paradigms. p. 285–309 *In*: R.J. Etches and A.M. Verrinder Gibbins (eds.) *Manipulation of the Avian Genome*. CRC Press. Boca Raton, FL.
- WAKENELL, P.S., M.M. MILLER, R.M. GOTO, W.J. GAUDERMAN, and W.E. BRILES. 1996. Association between the *Rfp-Y* haplotype and the incidence of Marek's disease in chickens. *Immunogenet.* **44**:242–245.
- WANG, N., R.N. SHOFFNER, J.S. OTIS, and K.M. CHENG. 1982. The induction of chromosomal structural changes in male chickens by the alkylating agents: triethylene melamine and ethyl methanesulfonate. *Mut. Res.* **96**:53–66.
- WATT, J.M., J.N. PETITTE, and R.J. ETCHES. 1993. Early development of the chick embryo. *J. Morph.* **214**:1–18.
- WIGHT, P.A. and W.G. SILLER. 1980. Pathology of deep pectoral myopathy of broilers. *Vet. Pathology* **17**:29–39.
- WIGHT, P.A., W.G. SILLER, and L. MARTINDALE. 1981. March gangrene: deep pectoral myopathy, Oregon disease, green muscle disease. *Am. J. Pathol.* **103**:159–161.
- WILSON, B.W. 1990. Developmental and maturational aspects of inherited avian myopathies. *Proc. Soc. Exp. Biol. Med.* **194**:87–96.
- WILSON, B.W., P.S. NIEBERG, R.J. BUHR, B.J. KELLY, and F.Z. SHULTZ. 1990. Turkey muscle growth and focal myopathy. *Poult. Sci.* **69**:1553–1562.
- WISHART, G.J. 1985. Quantitation of the fertilizing ability of fresh compared with frozen and thawed fowl spermatozoa. *Brit. Poult. Sci.* **26**:375–380.
- WOO, S.L., A. DUGAICZYK, M.J. TSAI, E.C. LAI, J.F. CATTERALL and B.W. O'MALLEY. 1978. The ovalbumin gene: cloning of the natural gene. *Proc. Natl. Acad. Soc. (USA)* **75**:3688–3692.
- WOODARD, A.E., H. ABPLANALP, J.M. PISENTI, and L.R. SNYDER. 1983. Inbreeding effects on reproductive traits in the ring-necked pheasant. *Poult. Sci.* **62**:1725–1730.
- WOOSTER, W.E., N.S. FECHHEIMER, and R.G. JAAP. 1977. Structural rearrangements of chromosomes in the domestic chicken: experimental production by X-irradiation of spermatozoa. *Can. J. Genet. Cytol.* **19**:437–446.
- ZARTMAN, D.L. 1971. X-ray-induced chromosomal aberrations in chickens. *Genetics* **68** (Suppl.):77.

Appendix 1. Instructions and survey form sent to researchers

INSTRUCTIONS FOR COMPLETING THE AVIAN GENETIC RESOURCES SURVEY

We appreciate your willingness to assist us in this survey. Below are some suggestions and clarifications for the enclosed survey form. If you need more space to complete your catalog of genetic stocks, please feel free to copy the survey form. We would also be interested in any relevant information you wish to attach that concerns your stocks (previously published summary information, references, expanded descriptions, etc.).

1. Curating researcher or person in charge of these stocks: this is the name of the person responsible for the poultry stocks recorded in the survey. This person can be contacted for further information on these stocks. If more than one person is responsible for a given stock or group of stocks, please write in the other names and addresses/phone numbers at the top of the page, and indicate the stocks that each person controls.

2. Name of Stock: how the stock is identified.

3. Species: chicken, turkey, coturnix quail or other avian species.

4. Stock Description/Characteristics: breed and any special stock characteristics, including degree of inbreeding (for inbred stocks), selection criteria, and/or mutations or chromosomal abnormalities present. If this stock is unchanged since it was listed in the 1988 International Registry, just write in "see 1988 registry" under this category. If you need more space to adequately describe a stock, please attach the expanded description to the survey form.

5. Eliminated? Date/Reason: if a stock has been eliminated, please indicate date of and reason for elimination (i.e., loss of funding, housing no longer available, stock no longer needed, etc.).

6. # Birds for Maint. (M & F): how many male and female birds are needed in a core flock to maintain a viable breeding population of each stock.

7. Funding Situation: security of the funding for that stock, at present and projected five years. If possible, please indicate source of funding: Government (NIH, NSF, USDA, etc.); Academic (university or institution, Hatch funds); Private (private organizations or individuals).

8. Other Loc.? (Y/N): is the stock maintained at one or more other institutions/locations? If yes, please give the name and address of the person(s) responsible for this stock at the other location(s), if available.

9. Cryo.? (Y/N): is the stock also (or only) kept as cryopreserved cells? If yes, please indicate the type of cells that have been cryopreserved (semen, blastodiscs, etc.), and where this material is kept.

SURVEY OF AVIAN GENETIC RESOURCES, 1996

Curating Researcher or person in charge of these stocks:

Address:

Phone Number: _____ **Fax:** _____ **Email:** _____

Name of Stock*	Species	Stock Description/Characterstics	Eliminated? Date/Reason	# Birds for Maint. (M & F)	Funding Situation	Other Loc? (Y/N)*	Cryo? (Y/N)*
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[illegible]

1. Do you make your stocks available to other researchers (for reproduction or research)?
2. Do you know of other researchers who maintain avian genetic stocks? If so, please write their name and address on the back of this form, or ask them to contact me. Thank you, Jacqueline M. Pisenti, GRCP, University of California, Davis, CA 95616, Fax (916) 754-8505, email: jmpisenti@ucdavis.edu

* please see instructions on the back of this form.

Appendix 2. Genetic stock collection data

THE INFORMATION PRESENTED HERE was collected through a survey initiated in 1995 by the University of California Genetic Resources Conservation Program. Many stocks developed before 1988 were listed in the International Registry of Poultry Genetic Stocks (SOMES, R.G., JR. 1988. International Registry of Poultry Genetic Stocks. Storrs Agr. Exp. Sta. Bull. 476. The University of Connecticut, Storrs). Therefore, curators listed in the Somes registry were the starting point for this survey. In addition, queries were sent to members of the NE-60 Regional Research Project (a USDA-organized group of researchers working on the genetic basis for resistance and immunity to avian diseases), academic and government members of the Poultry Science Association (source list: PSA Membership Directory, 1995-98) who indicated a focus on genetics or immunology, and individuals listed by the 1997 Poultry Science Resource List (prepared by the Poultry Science Association and R.D. Reynnells (USDA/CSREES)) who specialized in genetics or immunology and were affiliated with academic institutions with poultry, avian, or animal science departments.

Altogether, a total of more than 100 individuals were polled. Seventy-five responded and, of those, 40 currently maintain poultry genetic stocks as researcher-curators. These curators represent 27 different institutions (24 in the US, 3 in Canada). Approximately one-half of the institutions maintain 10 or fewer genetic stocks and only six maintain more than 20 stocks (University of Arkansas-Lafayette, University of California-Davis, University of Connecticut-Storrs, University of Wisconsin-Madison, USDA-ADOL East Lansing, MI, and University of British Columbia-Vancouver). About one-third of the reported stocks are considered to be at serious risk of loss in the near future, and at least 94 of the surveyed stocks were eliminated between

1995 and 1998. Over half of these discarded stocks were not represented in collections at other institutions and thus, are lost to science. Most stocks are maintained as living birds, but 38 chicken genetic stocks are maintained only in the form of cryopreserved semen or blastodermal cells.

The survey data are presented in three tables. Table 2.1 is a listing of the 361 reported stocks grouped first by species (chicken, Japanese quail, turkey, and waterfowl/gamebird), then by category of genetic stocks (the 28 categories used are listed in Table 2.0, then described be-

Table 2.0. Stock categories.

Bloodtype-Gene pool
Bloodtype-Major Histocompatibility Complex (MHC)
Bloodtype-MHC-Inbred
Chromosomal variant
Endogenous virus
Endogenous virus-Inbred
Inbred
Mutant-Color, eggshell
Mutant-Color, feather
Mutant-Developmental defect-Eye
Mutant-Developmental defect-Face/limb
Mutant-Developmental defect-Skin/feather
Mutant-Developmental defect-Spine/tail
Mutant-Gene pool
Mutant-Immunological defect
Mutant-Neurological defect
Mutant-Physiological defect
Mutant-Reproductive defect
Mutant-Uncategorized
Pure breed
Ranombred
Selected-Behavioral trait
Selected-Egg trait
Selected-Growth trait
Selected-Immune trait
Selected-Physiological trait
Transgenic
Uncategorized

low). Table 2.2 lists stocks grouped by institution and curator, species, and category. Finally, Table 2.3 lists curators and their contact information. Accordingly, the most important information necessary to look up a given genetic stock includes 1) the species and type of stock or 2) the institution or curator who keeps that type of stock.

Description of stock categories

Bloodtype refers to cell-surface antigens that have been characterized on red or white blood cells. **Bloodtype-Major Histocompatibility Complex (MHC)** consists of stocks with a defined MHC or B-haplotype, and may be defined for a variety of other erythrocyte alloantigens. **Bloodtype-MHC-Inbred** is congenic (genetically identical) to an established highly inbred line, except for the specified bloodtype. **Bloodtype-Gene pool** stocks contain a variety of different bloodtypes. Leukocyte cell surface antigens are also defined for one gene pool stock (NIU Male Breeder Alloantigen Reservoir).

Chromosomal variant refers to stocks with alterations in chromosome structure. These variations include: insertions, deletions, translocations, inversions, aneuploidy, and polysomy. Stocks with specific insertions (naturally occurring or as a result of genetic engineering) are listed under the Endogenous virus or Transgenic categories.

Endogenous virus (EV) stocks are characterized by specific viral insertions (one or more insertions of a defined endogenous virus), and **Endogenous virus-Inbred** stocks are congenic (genetically identical) to an established highly inbred line, except for the specified viral insertion.

Inbred stocks usually have a level of inbreeding exceeding 95% ($F > 0.95$), although one exception is UCD 001 (F is approximately 0.90). Other inbred stocks include congenic stocks developed by continued back-crossing to a given inbred stock. Such congenic stocks are categorized by the unique gene or gene complex isolated in the inbred background (e.g., Bloodtype-MHC-Inbred, Endogenous virus-Inbred, and Mutant-Developmental defect-Face/limb). In such stocks, the inbred ancestral stock is listed under "Origin and History".

Mutant stocks are classified into the following categories:

Mutant-Color, eggshell
Mutant-Color, feather
Mutant-Developmental defect-Eye
Mutant-Developmental defect-Face/limb
Mutant-Developmental defect-Skin/feather
Mutant-Developmental defect-Spine/tail
Mutant-Gene pool
Mutant-Immunological defect
Mutant-Neurological defect
Mutant-Physiological defect
Mutant-Reproductive defect
Mutant-Uncategorized

Color variation for feather and eggshell also occur in the pure breed and randombred categories, particularly among the different chicken breeds. Researchers interested in such variants should look up the color genetics for these breeds or discuss this with the pure breed stock curators.

Some developmental defect mutations may actually fit into more than one category (e.g., *wingless-2* affects face, limbs, feathers, and kidneys, but was listed by its effect on the face and limbs). Some of the mutations listed under neurological, physiological, and reproductive defects can also cause developmental defects. On the other hand, a few of the mutations or variants did not clearly fit into any of the above specific categories, so were classified as **Mutant-Uncategorized**. A close reading of the mutant descriptions is recommended.

Mutant-Gene pool refers to stocks that contain two or more mutations, which may or may not affect the same structures or systems. Some unique mutations are only maintained in gene-pool stocks, and so do not appear under the more specific mutant categories. However, brief descriptions of all mutations carried within that stock are listed for each of the pooled stocks.

The **Pure breed** category includes all stocks that are maintained as a closed flock of an identified breed. It is usually not highly inbred, selected, or otherwise characterized. Some overlap occurs between the Pure breed category and the Randombred category. However, if an otherwise Pure breed stock is commonly used as a control/standard for another stock, it will be listed as Randombred.

Randombred stocks are usually purebred or synthetic (mixed commercial) stocks that are kept in large, closed populations (100 birds or more).

Typically propagated with little or no selection of breeding stock each generation, the number of progeny from each male or female usually depends upon their reproductive success at the time eggs are collected to produce the next generation.

Stocks that have been selectively bred for a quantitative characteristic are termed **Selected**—along with the identified trait: **Selected-Behavior trait**, **Selected-Egg trait**, **Selected-Growth trait**, **Selected-Immune trait**, and **Selected-Physiological trait**. Each stock usually has an unselected control paired with it, and two divergently selected stocks (e.g., for high or low body weight at 6 weeks) from the same source may be grouped together here.

Transgenic stocks are the product of genetic engineering. Such stocks have successfully integrated the introduced genetic material into their chromosomes.

Finally there is a disparate group of stocks classified as **Uncategorized**, either for want of better descriptive information or because there may be too few representatives of a type to warrant an additional category.

While most stocks clearly belong to a single descriptive category, some logically could be classified in more than one. In such situations, the stock was listed under the category that most closely fit the stock description. The distribution of all listed stocks according to these categories is summarized in Chapter 5 of this report.

Notes on specific fields

Table 2.1 contains the fields described below.

Stock name is also used in Table 2.2.

Stock name: The designation used usually incorporates the name of the institution where the stock is housed or was developed, and the name that the curator assigned to the stock. In Table 2.2, the stock name is followed by the words ‘Cryo. only’, if that stock is only maintained by cryopreservation. This information is given in the **Status** field for Table 2.1 (see below).

Stock description: Only a brief description of the unique characteristics of the stocks is provided; for more information, the curator should be contacted.

Origin and history: Information in this field includes when a stock was established or acquired,

its current and historical genetic background (if available), and any other possibly useful background information. In particular, for stocks that are congenic, the specific inbred stock with which it is essentially identical is noted.

Breed: The source breed(s) or genetic background for stocks is noted here.

Genetic characteristics: For those stocks that are characterized by a distinct mutation or set of mutations, this field presents information on the pattern of inheritance (autosomal, sex-linked), penetrance and expressivity (dominant, co-dominant, recessive, incomplete penetrance, maternal effect, sex-limited), and effect on viability (lethal, semi-viable; if a mutation is fully viable, no notation is provided).

Allele symbol: Here the short notation for the mutant form of the gene is listed. If the symbol is in question or has not yet been officially assigned, the allele symbol is written in parentheses (i.e., (wgr) for the wing-reduced syndrome).

Status: This field provides a subjective evaluation of the potential for continued financial support of each stock, assessed by the curator of that stock. ‘Good’ indicates dependable institutional or government support for at least the next two to three years. ‘Fair’ indicates less certain support. ‘Poor’ describes a stock which is seriously at risk of elimination. When the specific source of funding support for the stock was provided, that information was noted: USDA, Industry, Institutional, State Agricultural Experiment Station (Exp. Sta.), Hatch funds (Hatch), research funds (regional), or other (misc.). In some cases, a stock is no longer maintained as living birds, only by germplasm cryopreservation. For such stocks, the notation in the status field is ‘No live birds’. Finally, some once-distinct mutant stocks are no longer carried as separate accessions, but were recently combined with another accession, and the name of the recipient stock is indicated in the status field (e.g., ‘Combined with UBC G’). For the purposes of Table 2.1 and this report, the original mutant is still counted as a separate accession.

Number of birds: When available, this is the number of birds (males and females) kept to maintain the stock.

Cryopreservation: If no germplasm has been cryopreserved for a given stock, the entry in this field is ‘No’. If germplasm has been cryopreserved,

then the cell type is indicated (semen, germ cells, blastodisc cells (for dissociated blastodiscs)). Stocks that are only maintained as cryopreserved germplasm can be identified by the remark 'No live birds' in the Status field. Curators can be contacted for details on recovery of these stocks and costs associated with the recovery.

Available: Many curators provide eggs or chicks to other researchers for collaborative projects; some will provide eggs or chicks for a fee (usually to offset bird maintenance costs), while other stocks may not be available because of proprietary concerns. Curators should be contacted for details.

Curator: The surname of the researcher in charge of the given stock is indicated here. Contact information for them is listed in Table 2.3, alphabetically, by surname.

Disclaimer

The University of California Genetic Resources Conservation Program and the Avian Genetic

Resources Task Force present this information as a service to those interested in poultry genetic stocks that can be found at academic and government institutions in the USA and Canada. No certification is implied for the stocks listed herein, either their status with regard to freedom from egg-borne disease agents, or their current availability (note that availability at the time the survey was conducted is indicated). Inquiries regarding particular stocks should be directed to the individual curator(s). In addition, readers are reminded that it is not possible to guarantee the authenticity or scientific accuracy of the information presented herein.

Those wishing to acquire any of these stocks or genetic material derived from these stocks are reminded that they must obtain the appropriate customs forms and documentation, and follow the protocols governing such shipments. Even transfer between different states in the US or provinces in Canada may require health certification or other documentation. In the US, the USDA Veterinary Services can provide some of this information.

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
<u>Bloodtype-MHC (cont.)</u>										
	NIU Female Breeder Parent Stock B2 MHC: B2; used as female parent in immunological challenges.	Derived from Ancona synthetic stocks homozygous for non-MHC system genes	Mixed Ancona		B2	Fair	N/A	No	With collaboration	Briles
	NIU Female Breeder Parent Stock B5 MHC: B5; used as female parent in immunological challenges.	Derived from Ancona synthetic stocks homozygous for non-MHC system genes	Mixed Ancona		B5	Fair	N/A	No	With collaboration	Briles
	RPRL-Cornell JM-N MHC: B21; resistant to Marek's disease virus strain JM.	Derived from Cornell JM randombred stock; specific pathogen-free	SCWL		B21	Good: USDA	6M/42F	Semen	Yes	Bacon
	RPRL-Cornell JM-P MHC: B19; susceptible to Marek's disease virus strain JM.	Derived from Cornell JM randombred stock; specific pathogen-free	SCWL		B19	Good: USDA	6M/42F	Semen	Yes	Bacon
	UNH 192 MHC: B19.	Derived from cross of SCWL and Ancona; kept as closed flock since 1988	SCWL X Ancona		B19	Good	10M/150F	No	Yes	Taylor
	<u>Bloodtype-MHC-Inbred</u>									
	ISU 19-13 Inbreeding coefficient (F)>0.98; MHC: B13.	Derived from crosses of 1920s ISU inbred stocks before 1935	SCWL		B13	Good	2M/16F	No	With collaboration	Lamont
	ISU 19-15.1 Inbreeding coefficient (F)>0.98; MHC: B15.1.	Derived from crosses of 1920s ISU inbred stocks before 1935	SCWL		B15.1	Good	2M/16F	No	With collaboration	Lamont
	ISU 8-15.1 Inbreeding coefficient (F)>0.98; MHC: B15.1.	Derived from crosses of 1920s ISU inbred stocks before 1935	Barred Leghorn		B15.1	Good	2M/16F	No	With collaboration	Lamont
	ISU G-B1 Inbreeding coefficient (F)>0.99; MHC: B13.	Congenetic with ISU GH	SCWL		B13	Good	3M/30F	No	With collaboration	Lamont
	ISU G-B2 Inbreeding coefficient (F)>0.99; MHC: B6.	Congenetic with ISU GH	SCWL		B6	Good	3M/30F	No	With collaboration	Lamont
	ISU GH-1 Inbreeding coefficient (F)>0.99; MHC: B1.	Derived from a cross of Ghostley Hatchery (MN) females and HN males in 1954	SCWL		B1	Good	2M/16F	No	With collaboration	Lamont
	ISU GH-13 Inbreeding coefficient (F)>0.99; MHC: B13.	Derived from a cross of Ghostley Hatchery (MN) females and HN males in 1954	SCWL		B13	Good	2M/16F	No	With collaboration	Lamont
	ISU GH-15.1 Inbreeding coefficient (F)>0.99; MHC: B15.1.	Derived from a cross of Ghostley Hatchery (MN) females and HN males in 1954	SCWL		B15.1	Good	2M/16F	No	With collaboration	Lamont
	ISU HN-12 Inbreeding coefficient (F)>0.99; MHC: B12.	Derived from a pure Kimber line from H&N in 1954	SCWL		B12	Good	2M/16F	No	With collaboration	Lamont
	ISU HN-15 Inbreeding coefficient (F)>0.99; MHC: B15.	Derived from a pure Kimber line from H&N in 1954	SCWL		B15	Good	2M/16F	No	With collaboration	Lamont
	ISU M15.2 Inbreeding coefficient (F)>0.98; MHC: B15.2; original stock thought to be resistant to lymphoid leukosis.	Imported from Egypt in 1954	Fayoumi		B15.2	Good	2M/16F	No	With collaboration	Lamont
	<u>Chicken genetic stocks</u>									

Appendix 2 Table 2.12

Table 2.1 Stock information									
Category	Stock name and description								
	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)									
Bloodtype-MHC-Inbred (cont.)									
ISU M5.1 Inbreeding coefficient (F)>0.98; MHC: B5.1; original stock thought to be resistant to lymphoid leukosis.	Imported from Egypt in 1954	Fayoumi		B5.1	Good	2M/16F	No	With collaboration	Lamont
ISU Sp21.2 Inbreeding coefficient (F)>0.98; MHC: B21.2.	Imported from Spain in 1954	Spanish		B21.2	Good	3M/30F	No	With collaboration	Lamont
NCSU GB-1 MHC: B13.	Acquired from Iowa State U; congenic with ISU GH	SCWL		B13	Good	40M/100F	No	Yes	Qureshi
NCSU GB-2 MHC: B6.	Acquired from Iowa State U; congenic with ISU GH	SCWL		B6	Good	40M/100F	No	Yes	Qureshi
RPRL 15.15-5 MHC: B5 from RPRL 1514.	Congenic with RPRL 1515; specific pathogen-free	SCWL		B5	Good: USDA	6M/35F	Semen	Yes	Bacon
RPRL 15.6-2 MHC: B2 from RPRL 611.	Congenic with RPRL 1515; specific pathogen-free	SCWL		B2	Good: USDA	6M/35F	Semen	Yes	Bacon
RPRL 15.7-2 MHC: B2 from RPRL 712.	Congenic with RPRL 1515; specific pathogen-free	SCWL		B2	Good: USDA	6M/35F	Semen	Yes	Bacon
RPRL 15.C-12 MHC: B12 from Reaseheath line C.	Congenic with RPRL 1515; specific pathogen-free	SCWL		B12	Good: USDA	6M/35F	Semen	Yes	Bacon
RPRL 15.N-21 MHC: B21 from Cornell JM-N.	Congenic with RPRL 1515; specific pathogen-free	SCWL		B21	Good: USDA	6M/35F	Semen	Yes	Bacon
RPRL 15.P-13 MHC: B13 from Cornell JM-P.	Congenic with RPRL 1515; specific pathogen-free	SCWL		B13	Good: USDA	6M/35F	Semen	Yes	Bacon
RPRL 15.P-19 MHC: B19 from Cornell JM-P.	Congenic with RPRL 1515; specific pathogen-free	SCWL		B19	Good: USDA	6M/35F	Semen	Yes	Bacon
RPRL 15I5 Inbreeding coefficient (F)>0.99; MHC: B15; endogenous viruses ev1, ev6, and ev10 or ev11; susceptible to avian leukosis virus A and B and Marek's disease virus.	Specific pathogen-free	SCWL		B15, ALVE1, ALVE6, ALVE10, ALVE11	Good: USDA	9M/63F	Semen	Yes	Bacon
RPRL 711 Inbreeding coefficient (F)>0.99; MHC: B2; endogenous viruses ev1 and ev3; susceptible to avian leukosis virus C and Marek's disease virus.	Specific pathogen-free	SCWL		B2, ALVE1, ALVE3	Good: USDA	12M/84F	Semen	Yes	Bacon
RPRL Line 0 MHC: B21; free of all known endogenous viruses; susceptible to avian leukosis viruses A, B & C.	Specific pathogen-free	SCWL		B21	Good: USDA	12M/84F	Semen	Yes	Bacon
UCD 003 Inbreeding coefficient (F)>0.99; A/E blood types 4/7; MHC: B17; shown to be resistant to Rous sarcoma and susceptible to Marek's disease virus .	Full-sib crosses since 1956; one of the reference lines in the East Lansing Chicken Genome Mapping Project, and forms the genetic background for a number of congenic strains that carry various MHC haplotypes or developmental mutations (see UCD stock list)	SCWL		B17, A4/E7	Poor	10M/12F	Semen	Yes	Abplanalp

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
<u>Bloodtype-MHC-Inbred (cont.)</u>										
UCD 253	MHC: B18; shown to be resistant to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003	SCWL		B18	Poor	5M/6F	Semen	Yes	Abplanalp
UCD 254	MHC: B15; shown to be susceptible to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003	SCWL		B15	Poor	5M/6F	Semen	Yes	Abplanalp
UCD 312	MHC: B24; shown to be susceptible to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003; MHC from New Hampshire chicken strain	SCWL		B24	Poor	5M/6F	Semen	Yes	Abplanalp
UCD 313	MHC: B3; shown to be resistant to Rous sarcoma virus.	Congenetic with UCD 003; died out as live birds in 1996	SCWL		B3	No live birds	0	Semen	With permission	Abplanalp
UCD 330	MHC: B21; shown to be resistant to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003; MHC from Australorp inbred line	SCWL		B21	Poor	5M/6F	Semen	Yes	Abplanalp
UCD 331	MHC: B2; shown to be resistant to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003; MHC from a dwarf SCWL	SCWL		B2	Poor	5M/6F	Semen	Yes	Abplanalp
UCD 335	MHC: B19; shown to be susceptible to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003; MHC from a commercial Richardson Mt. Hope SCWL	SCWL		B19	Poor	5M/6F	Semen	Yes	Abplanalp
UCD 336	MHC: BQ (similar to B21); shown to be resistant to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003; MHC from Red Jungle Fowl	SCWL		BQ	Poor	5M/6F	Semen	Yes	Abplanalp
UCD 342	MHC: BO; shown to be susceptible to Rous sarcoma virus.	Congenetic with UCD 003; MHC from a cross of Ceylon Jungle Fowl and Red Jungle Fowl	SCWL		BO	Poor	5M/6F	Semen	Yes	Abplanalp
UCD 361	A/E blood types 2/7.	Congenetic with UCD 003	SCWL		B17, A2/E7	Poor	5M/6F	No	Yes	Abplanalp
UCD 380	A/E blood types 4/7; resistant to Rous sarcoma.	Congenetic with UCD 003	SCWL		B17, A4/E7	Poor	5M/6F	No	Yes	Abplanalp
UCD 386	MHC: BR4/R4 (B15/B21 recombinant); shown to be resistant to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003	SCWL		BR4/R4	Poor	5M/6F	No	Yes	Abplanalp
UCD 387	MHC: BR5/R5 (B21/B15 recombinant); shown to be susceptible to Rous sarcoma and Marek's disease viruses.	Congenetic with UCD 003	SCWL		BR5/R5	Poor	5M/6F	No	Yes	Abplanalp
UNH 6.15-5	Inbreeding coefficient (F)>0.999; MHC: B5.	Congenetic with UNH 6-1	SCWL		B5	Good	10M/40F	No	Yes	Taylor
UNH 6.6-2	Inbreeding coefficient (F)>0.999; MHC: B2.	Congenetic with UNH 6-1	SCWL		B2	Good	10M/40F	No	Yes	Taylor
UNH-UCD 003	Inbreeding coefficient (F)>0.999; MHC: B17.	Acquired from U California-Davis in 1986	SCWL		B17	Good	10M/40F	No	Yes	Taylor
Chicken genetic stocks										
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Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Bloodtype-MHC-Inbred (cont.)										
	UNH-UCD 003.R1 MHC: <i>BF24-BG23</i> (B complex recombinant); backcrossed eight times to UCD-003.	Congenetic with UCD 003	SCWL		<i>BF24-BG23</i>	Good	10M/40F	No	Yes	Taylor
	UNH-UCD 003.R2 MHC: <i>BF2-BG23</i> (B complex recombinant); backcrossed eight times to UCD-003.	Congenetic with UCD 003	SCWL		<i>BF2-BG23</i>	Good	10M/40F	No	Yes	Taylor
	UNH-UCD 003.R3 MHC: <i>BF2-BG23</i> (B complex recombinant); backcrossed eight times to UCD-003.	Congenetic with UCD 003	SCWL		<i>BF2-BG23</i>	Good	10M/40F	No	Yes	Taylor
	UNH-UCD 003.R4 MHC: <i>BF2-BG23</i> (B complex recombinant); backcrossed eight times to UCD-003.	Congenetic with UCD 003	SCWL		<i>BF2-BG23</i>	Good	10M/40F	No	Yes	Taylor
	UNH-UCD 003.R5 MHC: <i>BF21-BG19</i> (B complex recombinant); backcrossed eight times to UCD-003.	Congenetic with UCD 003	SCWL		<i>BF21-BG19</i>	Good	10M/40F	No	Yes	Taylor
	UNH-UCD 003.R6 MHC: <i>BF21-BG23</i> (B complex recombinant); backcrossed eight times to UCD-003.	Congenetic with UCD 003	SCWL		<i>BF21-BG23</i>	Good	10M/40F	No	Yes	Taylor
	UNH-UCD 386 MHC: <i>BF15-BG21</i> (B complex recombinant).	Acquired from U California-Davis in 1995; congenic with UCD 003	SCWL		<i>BF15-BG21</i>	Good	10M/40F	No	Yes	Taylor
	UNH-UCD 387 MHC: <i>BF21-BG15</i> (B complex recombinant).	Acquired from U California-Davis in 1995; congenic with UCD 003	SCWL		<i>BF21-BG15</i>	Good	10M/40F	No	Yes	Taylor
	UNH-UCD 3C.1 MHC: <i>B17</i> , with recombined B complex.	Congenetic with UCD 003	SCWL		<i>B17</i>	Good	10M/40F	No	Yes	Taylor
	Chromosomal variant									
	Ottawa B-19/B-19 M13 MHC: <i>B19</i> ; segregating for DNA banding patterns using M13 phage.	Also referred to as Ottawa B19	SCWL		<i>B19</i>	No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa B-21/B-21 M13 MHC: <i>B21</i> ; segregating for DNA banding patterns using M13 phage.	Also referred to as Ottawa B21	SCWL		<i>B21</i>	No live birds	0	Blastodisc cells	With permission	Etches
	UCD-Cornell Mono-PNU Embryos homozygous for a large deletion on the MHC chromosome (16) lack most of the rDNA genes and die early in embryogenesis; heterozygotes develop normally and are viable.	Acquired from Cornell in 1995	SCWL			Good	120	No	With collaboration	Delany
	UCD-Cornell Trisomic Trisomy or tetrasomy of the MHC-containing chromosome (16); such aneuploids can hatch and reach maturity, but are often small and have poor production characteristics.	Acquired from Cornell in 1995	SCWL			Good	120	No	With collaboration	Delany
Chicken genetic stocks										
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Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Chromosomal variant (cont.)										
Wisconsin Chromosomal Rearrangements	Six chromosomal translocations and two inversions involving chromosomes 1 through 4 and Z.	Kept in genetic background of Wisconsin New Hampshire	New Hampshire			Fair: institutional	4M/6F	No	With collaboration	Bitgood
Endogenous virus	RPRL 15B1 Non-inbred; MHC: B5 and B15; susceptible to avian leukosis viruses A, B & C and Marek's disease virus; contains endogenous virus ev1.	Specific pathogen-free	SCWL		B5, B15, ALVE1	Good: USDA	6M/42F	Semen	Yes	Bacon
Endogenous virus-Inbred	RPRL 100B Inbreeding coefficient (F)>0.999; MHC: B2; endogenous viruses ev1 and ev3; susceptible to avian leukosis viruses B, C, and E, and Marek's disease virus.	Congenic with RPRL 712; specific pathogen-free	SCWL		B2, ALVE1, ALVE3	Good: USDA	6M/42F	Semen	Yes	Bacon
RPRL 613	Inbreeding coefficient (F)>0.999; MHC: B2; endogenous viruses ev1 and ev3; susceptible to avian leukosis viruses A,B, &C, and resistant to Marek's disease virus.	Specific pathogen-free	SCWL		B2, ALVE1, ALVE3	Good: USDA	12M/84F	Semen	Yes	Bacon
RPRL 712	Inbreeding coefficient (F)>0.999; MHC: B2; endogenous viruses ev1 and ev3; susceptible to avian leukosis virus C and Marek's disease virus.	Specific pathogen-free	SCWL		B2, ALVE1, ALVE3	Good: USDA	12M/84F	Semen	Yes	Bacon
RPRL Reaseheath Line C	Inbreeding coefficient (F)>0.999; MHC: B12; endogenous viruses ev1, ev7, ev10; susceptible to leukosis B and C.	Specific pathogen-free	SCWL		B12, ALVE1, ALVE7, ALVE10	Good: USDA	6M/42F	Semen	Yes	Bacon
Inbred	ADOL 6C.7A ReCon One of 19 different recombinant lines (primarily congenic with RPRL 613), each subline with 12.5% of genome from RPRL 712 (sublines identified by letters).	12% RPRL 712, 88% RPRL 613	SCWL		B2	Good: USDA	4M/10F	No	Yes	Bacon
ADOL 6C.7B ReCon	One of 19 different recombinant lines (primarily congenic with RPRL 613), each subline with 12.5% of genome from RPRL 712 (sublines identified by letters).	12% RPRL 712, 88% RPRL 613	SCWL		B2	Good: USDA	4M/10F	No	Yes	Bacon
ADOL 6C.7C ReCon	One of 19 different recombinant lines (primarily congenic with RPRL 613), each subline with 12.5% of genome from RPRL 712 (sublines identified by letters).	12% RPRL 712, 88% RPRL 613	SCWL		B2	Good: USDA	4M/10F	No	Yes	Bacon
ADOL 6C.7D ReCon	One of 19 different recombinant lines (primarily congenic with RPRL 613), each subline with 12.5% of genome from RPRL 712 (sublines identified by letters).	12% RPRL 712, 88% RPRL 613	SCWL		B2	Good: USDA	4M/10F	No	Yes	Bacon
Chicken genetic stocks										
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Table 2.1 Stock information							
Category							
Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.
Available	Curator						
Chicken (cont.)							
Inbred (cont.)							
ADOL 6C.7F ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7G ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7I ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7J ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7K ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7L ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7M ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7N ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7P ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7R ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon
ADOL 6C.7S ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No
					Yes		Bacon

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Inbred (cont.)										
	ADOL 6C.7T ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No	Yes	Bacon
	ADOL 6C.7V ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No	Yes	Bacon
	ADOL 6C.7W ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No	Yes	Bacon
	ADOL 6C.7X ReCon One of 19 different recombinant lines (primarily congenic with RPRL 6I3), each subline with 12.5% of genome from RPRL 7I2 (sublines identified by letters).	12% RPRL 7I2, 88% RPRL 6I3	SCWL		B2	Good: USDA	4M/10F	No	Yes	Bacon
	Ottawa GF Inbreeding coefficient (F)>0.8.	Derived from Ottawa 3	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa GH Inbreeding coefficient (F)>0.8.	Derived from Ottawa 3	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa M2 Inbreeding coefficient (F)>0.7.	Derived from Ottawa 4	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa WG Inbreeding coefficient (F)>0.7.	Derived from Ottawa 8	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa XP Inbreeding coefficient (F)>0.7.	Derived from Ottawa 9	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
	UCD 001 Inbreeding coefficient (F)>0.8; from wild-type jungle fowl.	One of the reference lines used in the East Lansing Chicken Genome Mapping Project	Red Jungle Fowl			Good	50M/50F	No	With collaboration	Delany
	Wisconsin Ancona Inbreeding coefficient (F)>0.9; half-sibling inbreeding since the mid-1940s.	Kept as closed flock for more than 20 generations	Ancona			Fair: institutional	4M/15F	No	With collaboration	Bitgood
	Wisconsin Leghorn line UW-Sp2 Inbreeding coefficient (F)>0.9; half-sibling inbreeding since the mid-1940s.	Derived from 1940/1950 Canadian Spruceleigh strains	SCWL			Fair: institutional	4M/15F	No	With collaboration	Bitgood
Mutant-Color, feather										
	Wisconsin Autosomal Albino Homozygotes have white feathers and red eyes.	Acquired from U Massachusetts-Amherst in 1997 (UMass Autosomal Albino)	SCWL	Autosomal recessive.	c ^a	Fair: institutional	4M/12F	No	With collaboration	Bitgood
Chicken genetic stocks										
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Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Mutant-Developmental defect-Eye										
	UBC RC <i>Retinal degeneration</i> homozygotes do not form rods or cones in the retina, causing blindness.	Found in Minnesota Rhode Island Red in 1980; backcrossed yearly to UBC-Minnesota Rhode Island Red	Rhode Island Red	Autosomal recessive.	<i>rc</i> or <i>rd</i>	Poor	12 M/40F	No	Yes	Cheng
	UMass Pink Eye <i>Pink-eye</i> homozygotes have pink eyes and a high incidence of cataracts; feather color is also diluted.	Also has dominant extension of melanin (E)	Mixed	Autosomal recessive.	<i>pk</i>	Dispersal in 1998	25	No	Yes	Smyth
	Wisconsin Blind/Cataract Homozygous chicks are blind at hatch, with visible cataracts.	Kept in genetic background of Wisconsin SCWL	SCWL	Autosomal recessive semi-viable.	<i>bc</i>	Fair: institutional	6M/12F	No	With collaboration	Bitgood
	Wisconsin Pink-eye Homozygotes have pink eyes and a high incidence of cataracts; feather color is diluted, even with dominant extension of melanin (E).	Acquired from U Massachusetts-Amherst in 1998	Mixed	Autosomal recessive.	<i>pk</i>	Poor	4M/12F	No	Yes	Bitgood
	Wisconsin Pop-eye Homozygotes have a conical protrusion of the cornea (keratoconus) first seen at 5 wk posthatch.	Kept in Wisconsin New Hampshire genetic background	New Hampshire	Sex-linked recessive.	<i>pop</i>	Fair: institutional	6M/12F	No	With collaboration	Bitgood
Mutant-Developmental defect-Face/limb										
	Storrs Chondrodystrophy Homozygotes have short upper beaks, shortened long bones, and bent tibiae.	Mutation reported in SCWL in 1942; kept at Storrs since late 1940s	SCWL	Autosomal recessive embryonic lethal.	<i>ch</i>	Poor	5M/19F	No	Yes	Pierro
	Storrs Creeper Heterozygotes have shortened limbs, and homozygotes die before hatch; stock also carries rose comb (R) and pillopody (leg feathering: <i>Pf</i>).	Held at Storrs since the 1920s; mutation present in a few exhibition breeds	Mixed Rose comb WL	R and Pt are autosomal dominants; cp is an autosomal incomplete dominant, lethal in homozygotes.	<i>Cp, R, Pt</i>	Poor	10M/39F	No	Yes	Pierro
	Storrs Diplopodia-3 Homozygotes display moderate preaxial polydactyly, dwarfing, exposed viscera, and shortened upper beak.	Mutation reported in inbred SCWL in 1972; acquired from U Saskatchewan in the 1960s	Mixed SCWL	Autosomal recessive embryonic lethal.	<i>dp-3</i>	Poor	5M/22F	No	Yes	Pierro
	Storrs Diplopodia-5 Homozygotes display moderate preaxial polydactyly, dwarfing, exposed viscera, and shortened upper beak.	Mutation reported in 1983; acquired from U Saskatchewan in late 1980s	Mixed SCWL	Autosomal recessive embryonic lethal.	<i>dp-5</i>	Poor	5M/21F	No	Yes	Pierro
	Storrs Limbless Homozygotes do not form limb buds or limbs, and usually have a shortened upper beak.	Mutation reported in 1979 in a flock of Rhode Island Reds; acquired from U Wisconsin in late 1980s	Mixed SCWL	Autosomal recessive embryonic lethal.	<i>ll</i>	Poor	5M/20F	No	Yes	Pierro
	Storrs Micromelia-Abbott Homozygotes have distinctly shortened long bones.	Mutation reported in crooked-neck dwarf stock from U California-Davis	Mixed SCWL	Autosomal recessive embryonic lethal.	<i>mm-A</i>	Poor	5M/17F	No	Yes	Pierro
	Storrs Micromelia-Hays Homozygotes have distinctly shortened long bones.	Mutation reported in Rhode Island Red stock in 1944; held at Storrs since 1950s	SCWL	Autosomal recessive embryonic lethal.	<i>mm-H</i>	Poor	5M/20F	No	Yes	Pierro
	Storrs Nanomelia Homozygotes have severely shortened long bones in the limbs; may also carry the mutant allele for leg-feathering (<i>Pf</i>).	Mutation reported in SCWL stock by Landauer in 1965; held at Storrs since then; also has pillopody	Mixed SCWL	Autosomal recessive embryonic lethal.	<i>nm, Pt</i>	Poor	5M/20F	No	Yes	Pierro
Chicken genetic stocks										

Appendix 2 Table 2.19

Table 2.1 Stock information

Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Mutant-Developmental defect-Face/limb (cont.)										
Storrs Perocephaly	Homozygotes show variable effects; from abnormal upper beaks to microcephaly, synophthalmia, and cyclopia.	Mutation reported in flock of SCWL by Landauer in 1956; held at Storrs since then	Mixed SCWL	Autosomal recessive embryonic lethal with low penetrance.	<i>per</i>	Poor	5M/22F	No	Yes	Pierro
Storrs Polydactyly	Homozygotes and heterozygotes may have one or two extra preaxial toes, or a longer-than-normal first digit.	Mutation long known in exhibition stocks and reported in several ancient breeds	Mixed SCWL	Autosomal incomplete dominant.	<i>Po</i>	Poor	5M/22F	No	Yes	Pierro
Storrs Talpid-2	Homozygotes display extreme preaxial polydactyly (up to 10 digits per limb), overall dwarfing, exposed viscera, and cleft palate with a parrot-like upper beak.	Mutation reported in SCWL stock in 1959; acquired from U California Davis in 1996	SCWL	Autosomal recessive embryonic lethal.	<i>ta-2</i>	Poor	2M/10F	No	Yes	Pierro
Storrs Wingless-2	Homozygotes lack wings, have reduced legs, balloon-like feathers, short upper beak, cleft palate and small kidneys.	Mutation reported in flock of SCWL by Landauer in 1956; held at Storrs since then	SCWL	Autosomal recessive embryonic lethal.	<i>wg-2</i>	Poor	5M/22F	No	Yes	Pierro
UCD Cleft Primary Palate/Scaleless	Cleft <i>primary palate</i> (<i>cpp</i>) homozygotes lack most of the upper beak, may also have limb reductions if homozygous for <i>scaleless</i> ; for <i>scaleless</i> (<i>sc</i>): <i>scaleless</i> homozygotes do not form spurs, scales or most feathers.	Derived from crosses of: SCWL, UCD Scaleless-High and UCD Scaleless-Low; <i>cpp</i> was originally called <i>ectrodactyly</i> when found in UCD Scaleless stocks in 1966	Mixed	Autosomal recessive embryonic lethal (<i>cpp</i>); autosomal recessive semi-viable (<i>sc</i>).	<i>cpp, sc</i>	Poor	5M/5F	Semen	Yes	Abbott
UCD Coloboma X 003	Hemizygous females are moderately to severely dwarfed with mildly to severely cleft palates; some are lacking preaxial digits or have truncated wings and legs; some are edemic; the expression may be highly variable, even with the same parents.	Congenetic with UCD 003; mutation first reported in 1970 at U California-Davis	SCWL	Autosomal recessive embryonic lethal.	<i>cm</i>	Poor	5M	Semen	Yes	Abbott
UCD Diplopodia-1 X 003	Homozygotes display moderate preaxial polydactyly, dwarfing, exposed viscera, and shortened upper beak.	Near-congenetic with UCD 003; mutation first reported in 1947 at U California-Berkeley	SCWL	Autosomal recessive embryonic lethal.	<i>dp-1</i>	Poor	5M/5F	Semen	Yes	Abbott
UCD Diplopodia-3 X 003	Homozygotes display moderate preaxial polydactyly, dwarfing, some with exposed viscera, occasional cleft palate, and shortened upper beak.	Near-congenetic with UCD 003; mutation first reported in 1972 at U California-Berkeley	SCWL	Autosomal recessive embryonic lethal.	<i>dp-3</i>	Poor	5M/5F	No	Yes	Abbott
UCD Donald-duck Beak/Scaleless	<i>Donald duck</i> beak homozygotes have upward curled upper beaks, may have downward curled lower beaks; some hatch, but do not survive long; no known interaction with <i>scaleless</i> ; for <i>sc</i> , see: UCD Scaleless-Low; may also carry cleft <i>primary palate</i> (<i>cpp</i>), see: UCD Cleft Primary Palate/Scaleless.	Reported in 1967 in New Hampshire-type chickens at U California-Davis	Mixed	Autosomal recessive embryonic lethals (<i>cpp</i> and <i>dd-2</i>); autosomal recessive semi-viable (<i>sc</i>).	<i>dd-2, sc</i> , may also include <i>cpp</i>	Poor	4M/3F	Semen	Yes	Abbott

Table 2.1 Stock information

Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserved	Available	Curator
Chicken (cont.)										
Mutant-Developmental defect-Face/limb (cont.)										
Chicken	UCD Eudiplopodia X 003 Homozygotes have extra digits on the dorsal surfaces of the limb buds, which develop into extra, bidorsal toes on the feet and occasional dorsal knobs or digits on the wings.	Congenitic with UCD 003; mutation first reported 1959 in SCWL flock at U California-Berkeley	SCWL	Autosomal recessive embryonic lethal.	eu	Poor	5M/5F	Semen	Yes	Abbott
	UCD Limbless X 003 Homozygotes do not form limb buds or limbs, and usually have a shortened upper beak.	Near-congenitic with UCD 003; mutation first reported in 1979 at U Wisconsin	SCWL	Autosomal recessive embryonic lethal.	//	Poor	5M/5F	No	Yes	Abbott
	UCD Polydactyly X 003 Homozygous and heterozygous mutants may have one or two extra preaxial toes, or a longer-than-normal first digit.	Near-congenitic with UCD 003; mutant allele common in exhibition chickens	SCWL	Autosomal dominant with incomplete penetrance.	Po	Poor	3M/3F	Semen	Yes	Abbott
	UCD Stumpy X 003 Homozygotes have conical leg buds and a poorly to non-vascularized allantois that never gets larger than the head; embryo death, typically between the fifth and seventh day, is associated with massive multiple hemorrhages.	Congenitic with UCD 003; mutation reported at U California-Davis in 1966 in New Hampshire X Cornish	SCWL	Autosomal recessive embryonic lethal.	stu	Poor	5M/5F	Semen	Yes	Abbott
	UCD Talpid-2 X 003 Homozygotes display extreme preaxial polydactyly, dwarfing, exposed viscera, and cleft, parrot-like upper beak.	Congenitic with UCD 003; mutation first reported in 1959 at U California-Davis in a SCWL flock	SCWL	Autosomal recessive embryonic lethal.	ta-2	Poor	5M/5F	Semen	Yes	Abbott
	UCD Wing-reduced X 003 A complex syndrome; affected birds lack toenails and some phalanges (usually on digit 3, sometimes digit 4) and may have truncated wings.	Syndrome found in UCD 413 (muscular dystrophy) in 1990; crossed several times to UCD 003	SCWL	Multifactorial, not yet fully defined.		Poor	3M/3F	No	Yes	Abbott
	UCD Wingless-2 X 331 Homozygotes lack wings, have reduced legs, balloon-like feathers, short upper beak, cleft palate and small metanephric kidneys.	Congenitic with UCD 331; mutation first reported in 1956 at U Connecticut-Storrs	SCWL	Autosomal recessive embryonic lethal.	wg-2	Poor	5M/5F	Semen	Yes	Abbott
	Wisconsin Ametapodia Heterozygotes lack metatarsals in the legs and metacarpels in the wings; homozygotes usually die early in development.	Acquired from U Massachusetts-Amherst in 1997 (UMass Ametapodia)	Mixed	Autosomal dominant, lethal in homozygotes.	Mp	Fair: institutional	4M/12F	No	With collaboration	Bitgood
	Wisconsin Limbless Homozygotes do not form limb buds or limbs, and sometimes have a short upper beak.	Kept in Wisconsin Leghorn genetic background	SCWL	Autosomal recessive embryonic lethal.	//	Fair: institutional	6M/20F	No	With collaboration	Bitgood
	Wisconsin Talpid-2 Homozygotes display extreme preaxial polydactyly, dwarfing, exposed viscera, and cleft, parrot-like upper beak.	Mutation first reported in 1959 at U California-Davis; acquired in 1990s; kept in Wisconsin Leghorn genetic background	SCWL	Autosomal recessive embryonic lethal.	ta-2	Fair: institutional	6M/12F	No	With collaboration	Bitgood
Wisconsin Wingless-2 Homozygotes lack wings, have reduced legs, balloon-like feathers, short upper beak, cleft palate and small kidneys.	Mutation first reported in 1956 at U Connecticut-Storrs; now kept in Wisconsin Leghorn genetic background	SCWL	Autosomal recessive embryonic lethal.	wg-2	Fair: institutional	6M/12F	No	With collaboration	Bitgood	

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Mutant-Developmental defect-Skin/feather										
Storrs	Ottawa Naked	Mutation first reported by R. Crawford in 1982; acquired from U Saskatchewan in late 1980s	New Hampshire	Autosomal recessive.	nk	Poor	3M/17F	No	Yes	Pierro
Storrs	Ptilopody	Mutation common in exhibition breeds; introduced into Storrs Nanomelia with Buff Cochins cross; also in Storrs Creeper;	Mixed SCWL	Autosomal dominant with multifactorial modifiers.	Pt	Combined with Storrs Nanomelia and Creeper	see: Storrs Nanomelia	No	Yes	Pierro
Storrs	Scaleless	Mutation reported in New Hampshire-type stock in 1957; acquired from U California-Davis in 1965	Mixed New Hampshire	Autosomal recessive semi-viable.	sc	Poor	10M/30F	No	Yes	Pierro
UCD	Ichthyosis X 003	Near-congenic with UCD 003; mutation first reported in 1957 at U California-Berkeley	SCWL	Autosomal recessive semi-viable.	dehy	Poor	5M/5F	Semen	Yes	Abbott
UCD	Naked-neck	Mutation common in exhibition breeds and in some commercial strains	Mixed SCWL	Autosomal dominant.	Na	No live birds	0	Semen	With permission	Abbott
UCD	Scaleless-High	Mutation first reported at U California-Davis in 1957; originally non-viable, but crossbreeding and selection dramatically increased viability; background breeds include Comish, SCWL, Barred Rock, New Hampshire, Red Jungle Fowl	Mixed	Autosomal recessive semi-viable, with multifactorial modifiers.	sc	Poor	15M/15F	Semen	Yes	Abbott
UCD	Scaleless-Low	Mutation first reported at U California-Davis in 1957; originally non-viable, but crossbreeding and selection dramatically increased viability; background breeds include Comish, SCWL, Barred Rock, New Hampshire, Red Jungle Fowl	Mixed	Autosomal recessive semi-viable, with multifactorial modifiers.	sc	Poor	15M/15F	Semen	Yes	Abbott
Wisconsin	Tardy Feathering	Kept in genetic background of Wisconsin Leghorn crossed with Wisconsin New Hampshire	SCWL	Autosomal recessive.	t	Fair: institutional	6M/12F	No	With collaboration	Bitgood

Chicken genetic stocks

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Table 2.1 Stock information

Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Mutant-Developmental defect-Spine/tail										
Storrs Dominant Rumplessness Homozygotes lack pygostyle, caudal vertebrae, uropygial gland and tail feathers; fertility is usually low if naturally mated.		Mutation reported in 1925; held at Storrs since then	SCWL	Autosomal dominant.	<i>Rp</i>	Poor	6M/21F	No	Yes	Pierro
Storrs Recessive Rumpless Homozygotes have fused or absent pygostyle and caudal vertebrae, and some kyphoscoliosis with extra ribs.		Mutation reported in SCWL stock in 1945; held at Storrs since then; also has <i>pericephaly (per)</i> ; kept homozygous for <i>rp-2</i>	Mixed SCWL	Autosomal recessive with incomplete penetrance.	<i>rp-2</i>	Poor	10M/67F	No	Yes	Pierro
UCD Scoliosis Affected birds show moderate to severe kyphoscoliosis starting at 5 weeks, and clearly visible in x-rays at 12 weeks; males are more severely affected than females.		Multifactorial syndrome uncovered in the 1950s during inbreeding of stocks at U California-Berkeley	Mixed Rose comb WL	At least two (maybe three) autosomal recessive genes.		No live birds	0	Semen	With permission	Abbott
Mutant-Gene pool										
OSU Dwarf Leghorn Includes seven mutations: <i>sex-linked dwarf</i> ; four autosomal mutations that cause: <i>blastoderm degeneration (bld)</i> , <i>blood ring</i> and <i>early embryo death (blr)</i> , and semi-lethal <i>chick edema</i> (when hom. for <i>ed-a</i> and <i>ed-b</i>); and two new mutations that have not yet been reported: <i>transient congenital tremor</i> and <i>ectopia</i> .		Mutation kept in commercial SCWL background, but as closed flock for seven generations; developed by P. Bernier	SCWL	All recessive; one sex-linked viable (<i>dw</i>), four autosomal lethal or semi-lethal (<i>blr</i> , <i>bld</i> , <i>ed-a</i> , <i>ed-b</i>).	<i>dw</i> , <i>blr</i> , <i>bld</i> , <i>ed-a</i> , <i>ed-b</i>	Fair	20M/50F	No	Yes	Savage
UBC-Minnesota Marker Gene pool with 13 dominant mutations: <i>polydactyl</i> , <i>ptilopody</i> , <i>pea</i> and <i>rose comb</i> , <i>uropygial bifurcation</i> , <i>multiple spurs</i> , <i>naked neck</i> , <i>crest</i> , <i>silver</i> , <i>sex-linked barring</i> , <i>dominant white</i> , <i>muffs</i> and <i>beard</i> , and <i>blue egg shell</i> ; may also contain recessive mutations <i>creeper (Cp)</i> and <i>protoporphyrin inhibitor (pr)</i> .		Acquired from U Minnesota-St. Paul	Mixed	Gene pool of various dominant and recessive mutations; see description.	<i>Po</i> , <i>Pt</i> , <i>P</i> , <i>R</i> , <i>U</i> , <i>M</i> , <i>Na</i> , <i>Cr</i> , <i>S</i> , <i>B</i> , <i>I</i> , <i>Mb</i> , <i>O</i> , <i>Cp</i> , <i>pr</i>	Poor	8M/40F	No	Yes	Cheng
UCD Diplopodia-3/Scaleless High <i>Diplopodia-3 (dp-3)</i> mutants display moderate preaxial polydactyly, dwarfing, exposed viscera, occasional cleft palate, and shortened upper beak; homozygous scaleless mutants lack spurs, scales and most feathers.		A recent cross (1997) of UCD Scaleless-High with UCD Diplopodia-3 X 003	Mixed	Two autosomal recessives; one embryonic lethal (<i>dp3</i>), one semi-viable (<i>sc</i>).	<i>dp-3</i> , <i>sc</i>	Poor	3M/5F	Semen	Yes	Abbott
UCD Eudiplopodial/Limbless See UCD Eudiplopodia and UCD Limbless descriptions.		Mildly inbred, including some UCD 003; crossed with UCD Scaleless-High and -Low in 1996	SCWL	Two autosomal recessive embryonic lethals.	<i>eu</i> , <i>ll</i>	Poor	10M/10F	Semen	Yes	Abbott
UCD Limbless/Stumpy See UCD Limbless and UCD Stumpy descriptions.		Ancestral background includes leukosis-free stocks kept at U California-Davis in the 1980s	SCWL	Two autosomal recessive embryonic lethals.	<i>ll</i> , <i>stu</i>	Poor	4M/4F	Semen	Yes	Abbott
UCD Silkie cross UCD Silkie crossed to UCD-003 or UCD Scaleless-low to decrease inbreeding and enhance egg production, then repeatedly back-crossed to UCD Silkie (see UCD-Silkie for description of mutations).		Derived from UCD Silkie; in 1995 crossed with both UCD 003 and UCD Scaleless-Low to increase vigor; backcrossed repeatedly to UCD Silkie	Mixed Silkie	Five autosomal dom. (<i>Cr</i> , <i>Po</i> , <i>Pt</i> , <i>Fm</i> , <i>Mb</i>), one sex-linked rec. (<i>ld</i>), two autosomal rec. (<i>h</i> , <i>sc</i>).	<i>Cr</i> , <i>Fm</i> , <i>h</i> , <i>id</i> , <i>Mb</i> , <i>Po</i> , <i>Pt</i> , <i>sc</i>	Poor	4M/7F	No	Yes	Abbott

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Mutant-Gene pool (cont.)										
UCD Talpid-2/Wingless-2	See UCD Talpid-2 and UCD Wingless-2 descriptions.	Mildly inbred, including some UCD 003; crossed with UCD Scaleless-High and -Low in 1996	SCWL	Two autosomal recessive embryonic lethals.	ta-2, wg-2	Poor	10M/10F	Semen	Yes	Abbott
Mutant-Immunological defect										
Arkansas Smyth Line B101	Selected for high expression of delayed amelanogenesis (autoimmune vitiligo: increasing amount of white feathers with each molt) also: blindness and thyroiditis; amelanosis now seen >85% of offspring.	Acquired from J. Smyth at U Massachusetts-Amherst in 1996; also known as DAM line (delayed amelanogenesis)	Brown Leghorn, synthetic	Multifactorial with variable expressivity.		Good	10M/20F	No	With collaboration	Erf
Arkansas Smyth Line B102	Selected for high expression of delayed amelanogenesis (autoimmune vitiligo: increasing amount of white feathers with each molt) also: blindness and thyroiditis; amelanosis now seen >85% of offspring.	Acquired from J. Smyth at U Massachusetts-Amherst in 1998; also known as DAM line (delayed amelanogenesis)	Brown Leghorn, synthetic	Multifactorial with variable expressivity.		Good	10M/20F	No	With collaboration	Erf
Cornell Obese (B-13)	MHC B13; affected birds develop spontaneous autoimmune thyroiditis and, in females, a persistent right Mullerian duct.	Derived from Cornell C Strain, selected for MHC haplotype B13; closed flock for over 30 years; pedigreed annual reproduction	SCWL	Multifactorial, with as many as five major genes.	B13	Poor	40M/120F	No	Yes	Marsh
UCD 200	Inbreeding coefficient (F) ² >0.80; develops autoimmune scleroderma (progressive systemic sclerosis), with necrosis and sloughing of the combs and wattles (self-dubbing), polyarthritis, and some visceral lesions.	Mutation first reported at Oregon State U in 1961	SCWL	At least two recessive genes and multifactorial modifiers.	sd	Poor	10M/12F	No	Yes	Abplanalp
UMass Smyth Line	Delayed amelanosis (DAM) homozygotes display a gradual loss of feather pigment with successive molts (an autoimmune condition mimicking human vitiligo), with some retinal dystrophy and autoimmune thyroiditis.	Also know as the DAM line (for delayed amelanogenesis); mutation originally found in the UMass Brown Line	Brown Leghorn, synthetic	Multifactorial with variable expressivity.		Dispersal in 1998	200	No	Yes	Smyth
Mutant-Neurological defect										
Saskatchewan Epileptiform seizures	Homozygotes afflicted with seizures of varying severity throughout life of bird, but good viability with extra care; used as disease model in pharmacological and physiological research.	Synthetic strain, mostly Fayoumi, derived from Agriculture Canada (CFAR) stocks in 1963	Mixed Fayoumi	Autosomal recessive semi-viable mutation with incomplete penetrance.	epi	Good	100M/170F	No	With collaboration	Classen
UNL Paroxysm	Post-hatch lethal mutation, causing seizures, slow growth and eventual death of hemizygous females.	Acquired from R. Cole at Cornell U in the 1960s	SCWL	Sex-linked recessive.	px	Fair: Hatch, regional, misc.	5M	No	With collaboration	Beck
Wisconsin Pirouette	Homozygous chicks expressing this neurological mutation will spin in circles for short periods.	Kept in Wisconsin Leghorn genetic background	SCWL	Autosomal recessive semi-viable.	pir	Fair: institutional	6M/12F	No	With collaboration	Bitgood
Chicken genetic stocks										

Appendix 2 Table 2.114

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
<u>Mutant-Physiological defect</u>										
Athens-Canadian Dwarfs	Sex-linked dwarf mutation in Athens-Canadian background.	Derived from Athens-Canadian Rando mbred	Commercial meat sire cross	Sex-linked recessive.	dw	Good	25M/50F	No	Yes	Burke
		Derived from the old Kemper SCWL strain, which is also was ancestral to Cornell K; pedigreed yearly reproduction	SCWL	Sex-linked recessive.	Dw, B15	Fair	120F	No	With collaboration	Marsh
		Derived from Storrs Muscular Dystrophy after an outcross with commercial SCWL in 1970s	Mixed SCWL	Not yet defined.		Good	50M/50F	No	With collaboration	Vellenan
		Mutation first reported in 1956 at U California-Davis; acquired from U Connecticut-Storrs in 1997; kept as homozygotes	Mixed	Autosomal recessive.	am	Good	50M/50F	No	With collaboration	Vellenan
		Mutation discovered in New Hampshire-type chicken stock in 1945; acquired from U California-Davis in 1950s	Mixed New Hampshire	Autosomal recessive embryonic lethal.	cn	Poor	5M/22F	No	Yes	Pierro
Storrs Crooked-neck Dwarf	Homozygotes are edemic and dwarfed, with crooked, fragile necks, thin amuscular legs, and no voluntary muscle contractions.	Mutation first reported in 1956 at U California-Davis; acquired 1958; kept as homozygotes	Mixed SCWL	Autosomal recessive.	am	Poor	5M/15F	No	Yes	Pierro
		Imported from Switzerland in 1987; full-sib inbreeding since 1968	SCWL	Multifactorial	B19	Poor	5M/6F	No	Yes	Abplanalp
UCD 077	Inbreeding coefficient (F)>0.95; MHC: B19; high blood lipid levels.	Derived from commercial meat stock displaying muscular dystrophy; closed flock since 1972	New Hampshire	Autosomal recessive.	am	Fair	15W/20F	No	Yes	Wilson
UCD 413	Muscular dystrophy homozygotes develop joint stiffness and pectoral muscle weakness starting about 4 weeks post-hatch, a condition produced by the replacement of these muscles by fat and connective tissue; some wing reduction defects not related to MD also seen.	Acquired from U Connecticut-Storrs in 1991; crossed to UCD 003 several times since 1996	Mixed SCWL	Autosomal recessive embryonic lethal.	cn	Fair	15W/20F	No	With permission	Abbott
UCD Crooked-neck Dwarf	Homozygotes are edemic and dwarfed, with crooked, fragile necks, thin amuscular legs, and no voluntary muscle contractions.	Out-crossed to UCD 003 in 1996; acquired from Pennsylvania State U in 1985	SCWL	Autosomal recessive, maternal-effect lethal.	rd	Poor	5M/5F	Semen	Yes	Abbott
UCD Riboflavin Transfer Deficient	Homozygotes can only make defective riboflavin binding protein; birds are viable, but such hens cannot put enough riboflavin into their egg albumen to support an embryo beyond 16 days of incubation; such embryos are dwarfed and hemorrhagic.									
Chicken genetic stocks										

Appendix 2 Table 2.115

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
<u>Mutant-Physiological defect (cont.)</u>										
	UCD-Cornell Autosomal Dwarf Homozygotes' body size reduced by 30% , recognizable by six to eight weeks; avg weight of female is 1173 g, egg weight 57.7g, 245 eggs/cycle, generally good viability but poor hatchability.	Mutation first reported in Cornell K Strain in 1973; transferred to U California-Davis in 1998	SCWL	Autosomal recessive.	<i>adw</i>	Poor	25M/50F	No	With collaboration	Delany
	Udel Riboflavin Transfer Deficient Homozygotes can only make defective riboflavin binding protein; birds are viable, but such hens cannot put enough riboflavin into their egg albumen to support an embryo beyond 16 days of incubation; such embryos die dwarfed and hemorrhagic.	Acquired from E. Buss at Pennsylvania State U	SCWL	Autosomal recessive, maternal-effect lethal.	<i>rd</i>	Poor	10M/50F	No	Yes	White
<u>Mutant-Reproductive defect</u>										
	Arkansas Sd Line Males homozygous for sperm degeneration have defective sperm due to efferent duct malfunction.	Mutation first reported in the 1960s at Ohio State in Delaware breed; out-crossed with SCWL from a single male in 1980s	Delaware X SCWL	Autosomal recessive, sex-limited.	<i>sdg</i> or <i>sd</i>	Good	25M/150F	No	No	Kirby
	Wisconsin Double Oviduct Line Both right and left oviduct form in most females of this line.	Rhode Island Red		Incompletely penetrant with multifactorial modifiers.	<i>Rov</i>	Fair: institutional	4M/15F	No	With collaboration	Bitgood
	Wisconsin Restricted Ovulator Hemizygous females lay few if any eggs, have high blood lipids, and produce no offspring; males do not appear to be affected.	Kept in Wisconsin Leghorn genetic background	SCWL	Sex-linked recessive.	<i>ro</i>	Fair: institutional	6M	No	With collaboration	Bitgood
<u>Mutant-Uncategorized</u>										
	ISU S1-19H Inbreeding coefficient (F)>0.5; MHC: <i>B19</i> ; <i>IrGAT^{high}</i> allele linked to MHC.	Derived from two inbred Hy-Line strains in 1964	SCWL		<i>B19</i> , <i>IrGAT^{high}</i>	Good	4M/24F	No	With collaboration	Lamont
	ISU S1-19L Inbreeding coefficient (F)>0.5; MHC: <i>B19</i> ; <i>IrGAT^{low}</i> allele linked to MHC.	Derived from two inbred Hy-Line strains in 1964	SCWL		<i>B19</i> , <i>IrGAT^{low}</i>	Good	4M/24F	No	With collaboration	Lamont
	ISU S1-11H Inbreeding coefficient (F)>0.5; MHC: <i>B1</i> ; <i>IrGAT^{high}</i> allele linked to MHC.	Derived from two inbred Hy-Line strains in 1964	SCWL		<i>B1</i> , <i>IrGAT^{high}</i>	Good	3M/30F	No	With collaboration	Lamont
	ISU S1-11L Inbreeding coefficient (F)>0.5; MHC: <i>B1</i> ; <i>IrGAT^{low}</i> allele linked to MHC.	Derived from two inbred Hy-Line strains in 1964	SCWL		<i>B1</i> , <i>IrGAT^{low}</i>	Good	3M/30F	No	With collaboration	Lamont
	Storrs Rose Comb Homozygotes and heterozygotes have a broad comb, covered with papillae, and a single spike at the back; has been associated with male infertility.	Mutation long known in exhibition stock; introduced into Storrs Creeper in 1960s	Mixed Rose comb WL	Autosomal dominant.	<i>R</i>	Combined with Storrs Creeper	see: Storrs Creeper	No	Yes	Pierro
Chicken genetic stocks										

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Mutant-Uncategorized (cont.)										
UCD Crest/Hemoglobin-D	<i>Hb^p</i> is a hemoglobin variant, found in both adult and embryonic hemoglobin, which is linked to the mutant crest allele. <i>Crest</i> causes a tuft of long feathers to form on the head.	Mutant form of hemoglobin reported linked to mutant <i>crest</i> allele, found at U California-Davis	SCWL	Linked autosomal dominant (<i>Cr</i>) and codominant (<i>Hb^p</i>).	<i>Cr</i> , <i>Hb^p</i>	No live birds	0	Semen	With permission	Abbott
UCD Multiplex Comb	Modifies the single-comb shape by causing points (fleshy projections) to form along accessory comb axes (up to five); these are usually smaller than the primary points of the single comb.	Causes conformational faults in comb shape of single-comb exhibition chickens	Mixed SCWL	Multifactorial.		No live birds	0	Semen	With permission	Abbott
Wisconsin Sex-linked Skin Color	Homozygous or hemizygous mutant birds cannot deposit yellow pigment in the skin.	Kept in Wisconsin Leghorn genetic background	SCWL	Sex-linked recessive.	<i>y</i>	Fair: institutional	6M/12F	No	With collaboration	Bitgood
Pure breed										
Arkansas Giant Jungle Fowl	Naturally highly resistant to Rous sarcoma virus; probably moderately inbred (foundation flock of 6 birds in 1950).	Imported from Southeast Asia in 1950 (one male and five females); kept as closed flock	Giant Jungle Fowl			Good	9M/45F	No	Yes	Anthony
Guelph Silkies	Breed with five major mutations: <i>Cr</i> (long feathers on crown of head), <i>Po</i> (one or more extra preaxial toes), <i>Pt</i> (legs and toes feathered), <i>Fm</i> (dark pigment in skin, internal organs), <i>Mb</i> (long feathers under chin), and <i>h</i> (feather defect: no hooklets).	Derived from exhibition stock; foundation was one pair; inbreeding minimized (about 35% in 1995)	Silkie	Five autosomal dominants (<i>Cr</i> , <i>Po</i> , <i>Pt</i> , <i>Fm</i> , <i>Mb</i>), one sex-link recessive (<i>td</i>), one autosomal recessive (<i>h</i>).	<i>Cr</i> , <i>Fm</i> , <i>h</i> , <i>td</i> , <i>Mb</i> , <i>Po</i> , <i>Pt</i>	Poor	20M/20F	No	With permission	Etches
NCSU Barred Plymouth Rock		Kept as closed flock used in research and teaching	Barred Plymouth Rock			Fair	4M/40F	No	Yes	Christensen
NCSU Rhode Island Red		Kept as closed flock used in research and teaching	Rhode Island Red			Fair	4M/40F	No	Yes	Christensen
Ottawa New Hampshire			New Hampshire			No live birds	0	Blastodisc cells	With permission	Etches
Saskatchewan Barred Plymouth Rock	Used as a slow-growing chicken model in cardiac and digestive physiology research, and as source of eggs with many blood and meat spots.	Acquired in 1920s	Barred Plymouth Rock			Good	20M/50F	No	With collaboration	Classen
Saskatchewan Brown Leghorn	Used as a slow-growing chicken model in cardiac and digestive physiology research, and as source of fertile eggs for use in research.	A strain of Danish Brown Leghorn acquired from Scattered Acres Hatchery in Hanover, Ontario in 1965	Brown Leghorn			Good	20M/50F	No	With collaboration	Classen
UBC-Minnesota Rhode Island Red	From a moderately inbred (F approx. 0.6) flock of Rhode Island Red. Body weight 4.5 lb.	Acquired from U Minnesota-St. Paul; kept as closed flock for over 20 generations; back-cross parent population for UBC RC	Rhode Island Red			Poor	8M/40F	No	Yes	Cheng
Chicken genetic stocks										

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Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Pure breed (cont.)										
	UCD Silkie Breed with five major mutations: <i>Cr</i> (long feathers on crown of head), <i>Po</i> (one or more extra preaxial toes), <i>Pt</i> (legs and toes feathered), <i>Fm</i> (dark pigment in skin, internal organs), <i>Mb</i> (long feathers under chin), and <i>h</i> (feather defect: no hooklets).	Descended from six exhibition birds acquired from a hobby-breeder in central California in 1994 (4M/2F)	Silkie	Five autosomal dom. (<i>Cr,Po,Pt,Fm,Mb</i>), one sex-linked rec. (<i>id</i>), one autosomal rec. (<i>h</i>).	<i>Cr, Fm, h, id, Mb, Po, Pt</i>	Fair	3M/3F	No	Yes	Abbott
	UI-Urbana Columbian Meat type, white/black feathers, slow growth, long legs, narrow breast; very mild selection for body size.	Non-exhibition (meat) variety; kept at U Illinois-Urbana as closed flock for over 30 years	Columbian			Good	50M/600F	No	With collaboration	Parsons
	UI-Urbana New Hampshire Meat type, moderate growth, good conformation; new bloodlines introduced approx. 1993 to increase body weight.	Non-exhibition (meat) variety; at U Illinois-Urbana for over 30 years; kept as closed flock except for outcross in 1993	New Hampshire			Good	50M/200F	No	With collaboration	Parsons
	UMass Light Brown Leghorn Light-brown Leghorn chicken that has never shown signs of delayed amelanogenesis.	Serves as control for UMass Smyth Line (no occurrence of delayed amelanogenesis)	Light Brown Leghorn			Dispersal in 1998	15M/35F	No	Yes	Smyth
Randombred										
	Arkansas Brown Line B101 MHC-matched control/parental line for Arkansas Smyth line B101.	Acquired from J. Smyth at U Massachusetts-Amherst in 1996; subline of ancestral parent stock for Smyth Line	Brown Leghorn		<i>e^b</i>	Good	10M/20F	No	With collaboration	Erf
	Arkansas Brown Line B102 MHC-matched control/parental line for Arkansas Smyth line B102.	Acquired from J. Smyth at U Massachusetts-Amherst in 1998; subline of ancestral parent stock for Smyth Line	Brown Leghorn, synthetic		<i>e^b</i>	Good	10M/20F	No	With collaboration	Erf
	Arkansas Light Brown Leghorn B101 MHC-matched control for Arkansas Smyth line B101; does not develop delayed amelanogenesis (autoimmune vitiligo).	Acquired from J. Smyth at U Massachusetts-Amherst in 1996	Light Brown Leghorn			Good	10M/20F	No	With collaboration	Erf
	Arkansas Randombred Randombred meat bird control from multiple parental strain cross.	Formed by crossing commercial meat (broiler) parent lines (seven male lines and eight female lines)	Commercial meat sire cross			Good	16M/48F	No	Yes	Anthony
	Athens Randombred Randombred control/standard; foundation males from commercial meat-type (WPR, WComish, NH); foundation females from egg-production selected RIR, BPR, WPR, NH, SCWL and Cornish from Southern Regional Experiment Stations.	Synthetic line integrating commercial and UGA Experiment Station meat-type stocks, started in 1956	Commercial meat sire cross			Good	30M/150F	No	Yes	Burke
	Athens-Canadian Randombred Randombred control/standard segregating for single, rose, and pea comb, with dominant white, some red and black feather color occurring.	Acquired from the Canadian Dept of Agriculture in 1958; includes White Wyandotte and three synthetic meat strains	Commercial meat sire cross		<i>R, P</i>	Good	60M/180F	No	Yes	Burke
Chicken genetic stocks										

Appendix 2 Table 2.118

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Randombred (cont.)										
	Cornell C Specials (B13) Control line for Cornell Obese; MHC B13; also can produce a persistent right Mullerian duct (often leading to the formation of two oviducts) in affected females.	MHC-matched to Cornell Obese; kept as closed flock for over 30 years; derived from Cornell C Strain	SCWL		B13	Poor	8M/42F	No	Yes	Marsh
	NCSU-Cornell K Strain MHC: B15.	Derived from Cornell K-strain	SCWL		B15	Good	40M/100F	No	Yes	Qureshi
	Ottawa 10 Control/standard maintained without selection since 1973.	No selection since 1973; derived from a combination of North American commercial egg-layer stocks	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa 20 Pedigreed randombred meat (broiler) reference stock from commercial sire lines.	Formed by combining nine commercial broiler sire stocks in 1979	Commercial meat sire cross			No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa 30 Pedigreed randombred meat (broiler) reference stock from commercial dam lines.	Formed by crossing nine commercial broiler dam stocks in 1979	Commercial meat sire cross			No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa 5 Control/standard maintained without selection since 1950.	No selection since 1950	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
	Ottawa 7 Control/standard maintained without selection since 1958.	Derived from four commercial egg-layer stocks and kept without selection since 1958; originally known as Kentville Control Strain 7	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
	UCD 412 Control/standard for UCD-413 (muscular dystrophy).	Kept as closed flock since 1972	New Hampshire			Fair	15M/20F	No	Yes	Wilson
	UMass Brown Line Brown homozygotes are solid brown as chicks; adult males are wild-type, females are stippled dark brown; serves as MHC-matched controls for UMass Smyth Line.	Source of delayed amelanogenesis mutation; serves as MHC-matched control for UMass Smyth Line	Brown Leghorn, synthetic	Autosomal recessive.	e ^b	Dispersal in 1998	90	No	Yes	Smyth
	Wisconsin Leghorn Closed population of Single-comb White Leghorns.	Kept as closed flock for over 30 generations; used as genetic background for several mutant stocks at U Wisconsin-Madison	SCWL			Fair: institutional	20M/180F	No	With collaboration	Bitgood
Wisconsin Leghorn line UW-6X Closed randombred/control population of 1950s origin egg-type commercial Single-comb White Leghorns.	From cross of commercial egg-layers (Spruceleigh X HN) in 1950s; kept as closed flock for over 20 years; almost extinct, only a single, low production female in 1998	SCWL			Poor	Few	No	With collaboration	Bitgood	
Wisconsin New Hampshire Closed randombred/control population of purebred New Hampshire chickens.	Kept as closed flock for over 30 generations; used as genetic background for several mutant stocks at U Wisconsin-Madison	New Hampshire			Fair: institutional	20M/180F	No	With collaboration	Bitgood	
Chicken genetic stocks										

Appendix 2 Table 2.119

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserved.	Available	Curator
Chicken (cont.)										
Selected-Behavioral trait										
Purdue KGB	Selected for non-aggressive behavior and resistance to stress ("Kinder, Gentler Bird").	Derived from a seven-way, wide commercial cross at the North Central Regional Poultry Laboratory in 1972	SCWL			Fair	100M/100F	No	With collaboration	Muir
		Derived from Purdue KGB	SCWL			Fair	150M/250F	No	With collaboration	Muir
Purdue MBB	Selected for more aggressive behavior ("Mean, Bad Bird").									
Selected-Egg trait										
Cornell K Strain	MHC: B15; resistant to leukosis complex (particularly Marek's disease) by selection after natural exposure to the viruses; good egg production characteristics.	Kept as closed flock since 1954; selected to optimize egg production, egg size, body weight, other economic egg traits until 1971, then randombred with minimal selection	SCWL		B15	Poor	165	No	Yes, fee	Dietert
		Derived from commercial stock acquired in 1991	Barred Plymouth Rock			Good	30M/130F	No	With permission	Etches
Guelph Barred Plymouth Rock	Commercial stock selected for egg production.	Derived from commercial stock acquired in 1991	SCWL			Good	30M/130F	No	With permission	Etches
		Derived from Ottawa 5 in 1950; divided into Ottawa 1 and 3 in 1971	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 1	Selected for high egg production and related traits.	Derived from seven Canadian ROP (egg-type) stocks in 1951; divided in 1969 into Ottawa 2 and 4	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
		Established from Ottawa 5 in 1950; divided into Ottawa 1 and 3 in 1971	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 3	Selected for high egg production and related traits.	Derived from seven Canadian ROP (egg-type) stocks in 1951; divided in 1969 into Ottawa 2 and 4	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
		Derived from Ottawa 7 in 1969	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 4	Selected for high egg production and related traits.	Derived from Ottawa 7 in 1969	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
		Derived from Ottawa 7 in 1969	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 8	Selected for high egg production and related traits.	Derived from Ottawa 7 in 1969	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
		Derived from Ottawa 7 in 1969	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 9	Selected for high egg production and related traits.	Derived from Ottawa 7 in 1969	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
		Derived from Ottawa 7 in 1969	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
UCD 058	Inbreeding coefficient (F)>=0.8; selected for increased egg production.		SCWL			Poor	5M/6F	No	Yes	Abplanalp
			SCWL			Poor	5M/6F	No	Yes	Abplanalp
UCD 082	Inbreeding coefficient (F)>0.76; selected for egg production with relaxed selection in recent generations.		SCWL			Poor	5M/6F	No	Yes	Abplanalp
			SCWL			Poor	5M/6F	No	Yes	Abplanalp

Chicken genetic stocks

Appendix 2 Table 2.120

Table 2.1 Stock information

Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Selected-Growth trait										
Auburn Tibial Dyschondroplasia-High	One of two strains that were developed from a commercial meat sire line by divergent selection for tibial dyschondroplasia; incidence is 95% for birds in this line after 11 generations of selection.	Derived from a commercial meat sire line acquired in 1988	Commercial meat sire line			Good	15M/70F	No	Yes	Berry
		Derived from a commercial meat sire line acquired in 1988	Commercial meat sire line			Good	15M/70F	No	Yes	Berry
		Derived from Ottawa 20	Commercial meat sire cross			No live birds	0	Blastodisc cells	With permission	Etches
		Derived from Ottawa 20	Commercial meat sire cross			No live birds	0	Blastodisc cells	With permission	Etches
		Derived from Ottawa 20	Commercial meat sire cross			No live birds	0	Blastodisc cells	With permission	Etches
		Derived from Ottawa 30	Commercial meat sire cross			No live birds	0	Blastodisc cells	With permission	Etches
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	No	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	No	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
Auburn Tibial Dyschondroplasia-Low	One of two strains that were developed from a commercial meat sire line by divergent selection for tibial dyschondroplasia; incidence is 6% for birds in this line after 11 generations of selection.	Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
PSU-Athens RB 14-42L (LoK)	Selected for minimum growth between 14 and 42 days from a starting population of 350.	Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato
		Derived from Athens-Canadian Randombred from U Georgia-Athens	Commercial meat stock X egg selected female lines			Good: Exp. Sta. & USDA	20M/60F	Semen, Germ cells	With collaboration	Barbato

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Selected-Growth trait (cont.)										
Virginia Body Weight-High	Selected 40 generations for high 8-week body weight.	Formed by crossing seven inbred lines kept at Virginia State in 1957	White Plymouth Rock			Poor	100M/100F	No	With collaboration	Siegel
		Formed by crossing seven inbred lines kept at Virginia State in 1957	White Plymouth Rock			Poor	100M/100F	No	With collaboration	Siegel
Selected-Immune trait										
Arkansas Progressor	Selected 20 generations for tumor growth (progression) following formation with exposure to Rous sarcoma virus.	Derived from SCWL maintained at U Arkansas since approximately 1945; thought to be highly inbred; also known as Arkansas Rous Sarcoma Progression	SCWL			Good	9M/45F	No	Yes	Erf
Arkansas Regressor	Selected 20 generations for tumor regression following formation with exposure to Rous sarcoma virus.	Derived from F-1 and F-2 progeny of crosses of SCWL and Giant Jungle Fowl; also known as Arkansas Rous Sarcoma Regression	SCWL X Giant Jungle Fowl			Good	9M/45F	No	Yes	Erf
Athens AR	Selected for resistance to aflatoxin.	Derived from a cross of Athens AR2.5 and AR3.0 in 1997	SCWL			Good	36M/36F	No	Yes	Burke
Auburn R	Resistant to coccidiosis; MHC: B3; other erythrocyte alloantigens: A/E2, C2, D3, H1, I2, K2, P3, "Q"2.	Kept as closed flock since 1948	SCWL		see description	Good	180	No	With collaboration	Ewald
Auburn S	Susceptible to coccidiosis; MHC: B5; other erythrocyte alloantigens: A/E2, C3, D2, H1, I2, K3, P2, "Q"1.	Kept as closed flock since 1952	SCWL		see description	Good	200	No	With collaboration	Ewald
Cornell S13	MHC: B13; highly susceptible to Marek's disease virus; free of most avian viruses.	Derived from Cornell S Strain; specific pathogen-free	SCWL		B13	Good	28M/106F	No	Yes, fee	Schat
Ottawa 2R	Selected for resistance to Marek's disease virus.	Derived from Ottawa-2 and-4; also referred to as Ottawa R2	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 3R	Selected for resistance to Marek's disease virus.	Derived from Ottawa-1 and-3; also referred to as Ottawa R3	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 8R	Selected for resistance to Marek's disease virus.	Derived from Ottawa 8 and-9; also referred to as Ottawa R8	SCWL			No live birds	0	Blastodisc cells	With permission	Etches
Virginia Antibody Line-High	Selected over 20 generations for high antibody response to sheep red blood cells.	Derived from a Cornell randombred SCWL, starting in 1977	SCWL			Poor	100M/100F	No	With collaboration	Siegel
Virginia Antibody Line-Low	Selected over 20 generations for low antibody response to sheep red blood cells.	Derived from a Cornell randombred SCWL, starting in 1977	SCWL			Poor	100M/100F	No	With collaboration	Siegel
Selected-Physiological trait										
Arkansas Ascites Resistant	Selected three generations for resistance to ascites under high altitude simulation; family selection.	Pedigreed commercial synthetic stocks	Commercial meat sire cross			Good	16M/48F	No	No	Anthony

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Chicken (cont.)										
Selected-Physiological trait (cont.)										
Arkansas Ascites Susceptible	Selected three generations for susceptibility to ascites under high altitude simulation; family selection.	Pedigreed commercial synthetic stocks	Commercial meat sire cross			Good	16M/48F	No	No	Anthony
Transgenic										
ADOL Trans-ALVE6	Homozygous for the <i>alv-6</i> transgene; expresses env glycoprotein of ALV subgroup A; resists infection with ALV subgroup A.		SCWL		ALVE6	Good: USDA	N/A	No	Yes	Bacon
Ottawa TR	Has incorporated the <i>alv-6</i> transgene.		SCWL		ALVE6	No live birds	0	Blastodisc cells	With permission	Etches
Uncategorized										
Ottawa 6	Not reported.		Not reported			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 80	Not reported.		Not reported			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa 90	Not reported.		Not reported			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa N3	Not reported.		Not reported			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa N4	Not reported.		Not reported			No live birds	0	Blastodisc cells	With permission	Etches
Ottawa N8	Not reported.		Not reported			No live birds	0	Blastodisc cells	With permission	Etches

Chicken genetic stocks

Appendix 2 Table 2.123

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Japanese quail										
Inbred										
UBC I	Full-sibling inbreeding for four generations, starting in 1992; relaxed inbreeding pressure in 1996 (crossing half sibs or less closely related birds).	Inbred starting in 1992, with relaxation of inbreeding for the last few years				Poor	8M/16F	No	Yes	Cheng
Mutant-Color, eggshell										
UBC CE	Females homozygous for <i>celadon</i> lay blue-shelled eggs.	Acquired from U Nagoya (Japan) in 1988; combined with UBC C in 1995		Autosomal recessive.	<i>ce</i>	Combined with UBC C in 1995	see: UBC C-CE	No	Yes	Cheng
UBC WE	Females homozygous for <i>white-egg</i> lay white-shelled eggs.	Cross of U Nagoya (Japan) WE and U Saskatchewan stock; combined with UBC W in 1995		Autosomal recessive.	<i>we</i>	Combined with UBC W in 1998	see: UBC W-WE	No	Yes	Cheng
Mutant-Color, feather										
Arkansas White	Homozygotes have pure white feathers with dark eyes, different from English White.	Mutation reported in Arkansas randombred Japanese quail		Autosomal recessive.		Poor	36M/36F	No		Anthony
Arkansas English White	Homozygotes have dark eyes and mostly white feathers with slashes of wild-type color.	Acquired from the Quail Resource Center		Autosomal recessive.	<i>wh</i>	Good	36M/36F	No		Anthony
UBC BH	<i>Black-at-hatch</i> homozygotes die by the sixth day of incubation; heterozygotes are viable, but have only faint remnants of the wild-type yellow stripes at hatch.	Acquired from U Nagoya (Japan) in 1988; frequently backcrossed to UBC A		Autosomal dominant.	<i>Bh</i>	Poor	6M/12F	No	Yes	Cheng
UBC C-CE	<i>Cinnamon</i> homozygotes have bright orange-brown feathers instead of medium brown, and eyes are red in the chick; females homozygous for <i>celadon</i> lay blue-shelled eggs.	<i>Cinnamon</i> mutation reported in UBC M in 1978; <i>celadon</i> mutation acquired from U Nagoya (Japan) in 1988; combined 1995		Autosomal recessives.	<i>cin, ce</i>	Poor	24M/48F	No	Yes	Cheng
UBC D	<i>Dark-eyed dilute</i> homozygotes have dark eyes and diluted feather color; homozygotes have unpigmented retinas and nearly white feathers; <i>albino</i> hemi- and homozygotes are nearly white.	Acquired in 1979, then combined with UBC AL (from U Saskatchewan) in 1984		Two allelic sex-linked recessives.	<i>al^p, al</i>	Poor	24M/48F	No	Yes	Cheng
UBC F-SB	<i>Fawn</i> heterozygotes have fawn-colored feathers, but <i>Yⁱ</i> is lethal in the homozygotes; <i>short-barb</i> homozygotes have feather barbs approximately 75% shorter than normal.	<i>Fawn</i> mutation from a breeder near Vancouver, BC in 1987; combined with UBC SB		Autosomal dominant <i>Yⁱ</i> is lethal in homozygotes; <i>sb</i> is an autosomal recessive.	<i>Yⁱ, sb</i>	Poor	24M/48F	No	Yes	Cheng
UBC H	<i>Extended brown</i> homozygotes and, to a lesser extent, heterozygotes, have extended distribution of black and dark brown pigment in the feathers, with both sexes appearing the same.	Back-crossed to UBC A each generation		Autosomal incomplete dominant.	<i>E</i>	Poor	6M/12F	No	Yes	Cheng

Table 2.1 Stock information

Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Japanese quail (cont.)										
Mutant-Color, feather (cont.)										
UBC RH	<i>Redhead</i> homozygotes have distinctly red head feathers (same as pansy); <i>ruffled</i> homozygotes have curved, bent or ruffled feathers.	Acquired from Virginia Polytechnic Inst. in 1977; combined with UBC RF in 1995		Autosomal recessive.	<i>e^{rh}</i>	Combined with UBC RF in 1995	see: UBC RF-RH	No	Yes	Cheng
UBC SI	<i>Silver</i> homozygotes have white feathers and a centrally depigmented retina (ring-retina); heterozygotes have greyish feathers, and may have a few white primaries.	Acquired from U Nagoya (Japan) in 1988; back-crossed to UBC A every generation		Autosomal incomplete dominant.	<i>B</i>	Poor	6M/12F	No	Yes	Cheng
UBC W-WE	<i>Recessive white</i> homozygotes have white feathers and dark eyes; heterozygotes have white in ventral feather tracts and face, with a mixture of dark and white feathers on the back; those homozygous for <i>we</i> lay white eggs.	Mutation <i>wh</i> from flock near Vancouver, BC (1976); <i>we</i> from a cross of stocks from U Nagoya and U Saskatchewan; combined 1995		Autosomal recessives.	<i>wh, we</i>	Poor	24M/48F	No	Yes	Cheng
UBC WB	<i>White-breasted</i> mutants have white feathers on face, ventral neck, breast, primaries and most secondaries.	Mutation reported in UBC A in 1975; combined with UBC PC in 1995		Autosomal recessive.	<i>wb</i>	Combined with UBC PC in 1995	see: UBC PC-WB	No	Yes	Cheng
UBC Y	<i>Yellow</i> heterozygotes have yellow (golden) feathers with restricted black pigment distribution; the mutation is lethal in homozygous form.	Mutation reported in UBC D in 1983; back-crossed to UBC A every generation		Autosomal dominant, lethal in homozygotes.	<i>Y</i>	Poor	6M/12F	No	Yes	Cheng
Mutant-Developmental defect-Skin/feather										
UBC DF-MDF	Two mutations acting in concert to produce defective feathers: short, sparse down at hatch, and defective barbs in later feathers; the <i>Df</i> dominant mutation is lethal in homozygotes.	Mutations reported in UBC W in 1979		Autosomal dominant (<i>Df</i>) and recessive (<i>mdf</i>) with variable expressivity.	<i>Df, mdf</i>	Poor	24M/48F	No	Yes	Cheng
UBC PC-WB	<i>Porcupine</i> mutants reproduce poorly and have feathers that resemble porcupine quills (the vanes do not uncoil); <i>white-breasted</i> mutants have white feathers on face, ventral neck, and breast; wing flight feathers (primaries and most secondaries) are also white.	<i>Pc</i> mutation reported in UBC W in 1979; <i>wb</i> mutation reported in UBC A in 1975; combined 1995		Autosomal recessives.	<i>pc, wb</i>	Poor	24M/48F	No	Yes	Cheng
UBC RF-RH	<i>Ruffled</i> homozygotes have curved, bent or ruffled feathers (<i>rf</i> mutation not yet reported in the literature).	<i>Rf</i> mutation reported in UBC B in 1983; <i>redhead</i> (<i>e^{rh}</i>) from Virginia Poly. Inst. in 1977; combined 1995		Autosomal recessives.	<i>rf, e^{rh}</i>	Poor	24M/48F	No	Yes	Cheng
UBC RT	<i>Rough-textured</i> homozygotes have feathers that appear matted and rough, and the females produce fewer viable embryos.	Mutation reported in UBC A in 1977; combined with UBC M in 1989		Autosomal recessive.	<i>rt</i>	Combined with UBC M in 1989	see: UBC M	No	Yes	Cheng
UBC SB	<i>Short distal barb</i> mutants show fusion in the distal barbs of contour feathers during feather growth; barbs are about 1/4 normal length, most visible in the contour feathers of the back.	Mutation reported in UBC A in 1982; pooled with UBC F		Autosomal recessive.	<i>sb</i>	Combined with UBC F	see: UBC F-SB	No	Yes	Cheng

Table 2.1 Stock information						
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status
						Number of birds
						Cryo-preserv.
						Available
						Curator
Japanese quail (cont.)						
Mutant-Developmental defect-Skin/feather (cont.)						
UBC SP <i>Spade</i> homozygotes have defective feathers (<i>sp</i> mutation not yet reported in the literature).	Mutation found in UBC AL in 1988; combined with UBC B in 1996			Autosomal recessive.	<i>sp</i>	Combined with UBC B in 1997
						see: UBC B
						No
						Yes
						Cheng
Mutant-Gene pool						
Wisconsin Japanese Quail Homozygous <i>wh</i> birds have white feathers; homozygous <i>we</i> females lay chalk-white eggs.	Acquired from U Massachusetts-Amherst in 1969			Autosomal recessive.	<i>wh, we</i>	Good: NASA
						150M/350F
						No
						Yes
						Wentworth
Mutant-Uncategorized						
UBC H5 Mutation causes H5 histones to form dimers in vitro.	Mutation reported in UBC WILD in 1992; crossed with UBC A in 1997			Incomplete dominant		Poor
						48M/96F
						No
						Yes
						Cheng
Randombred						
Arkansas RBC Randombred control strain from Eastern Shore Randombred.	Acquired from Eastern Shore as a randombred control in 1990					Good
						36M/36F
						No
						Anthony
Athens Control Quail Randombred control, with <i>white egg shell</i> mutation in the gene pool, propagated by random pair-matings.	Kept as closed flock since 1963					Good
						120M/120F
						No
						Yes
						Burke
Louisiana Randombred Quail Unselected, randombred population with some color mutations (<i>tuxedo, redhead, white egg</i>).	Kept at Louisiana State as closed flock for over 20 years					Good
						60M/120F
						No
						Yes
						Satterlee
Ohio R1 Propagated using 36 pairs each generation.	Derived from a cross of Athens Randombred, Athens white egg, and Wisconsin stock; kept as closed flock for 38 generations					Poor
						36M/36F
						No
						Yes
						Nestor
UBC A Randombred flock.	Derived from a cross of UCD Randombred quail and quail stock from Korea; kept as closed flock for over 70 generations					Poor
						48M/96F
						No
						Yes
						Cheng
UBC B Exceptionally nervous randombred; <i>spade</i> homozygotes have defective feathers.	Acquired from U Alberta in 1977, combined with UBC SP (<i>spade</i> mutation, affecting feathers) by 1998				<i>sp</i>	Poor
						24M/48F
						No
						Yes
						Cheng
UBC M <i>Rough-textured</i> homozygotes have feathers that appear matted and rough, and the females produce fewer viable embryos (<i>rt</i> mutation not yet reported in the literature); UBC M have the <i>extended brown</i> allele, and are slightly heavier than UBC A.	UBC M from a commercial (Marsh Farms) strain in 1975; combined with UBC RT in 1989			Autosomal recessive.	<i>rt</i>	Poor
						24M/48F
						No
						Yes
						Cheng
UBC N Very docile randombred.	Acquired from U Nagoya (Japan) in 1988					Poor
						24M/48F
						No
						Yes
						Cheng
Japanese quail genetic stocks						

Table 2.1 Stock information										
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Japanese quail (cont.)										
Randombred (cont.)										
UBC NC	Randombred that has proven to be very sensitive to changes in photoperiod.	Acquired from NCSU-randombred quail in 1990				Poor	24M/48F	No	Yes	Cheng
UBC S		Acquired from U Saskatchewan in 1983				Poor	24M/48F	No	Yes	Cheng
UBC WILD		Foundation stock consisted of 12 feral birds caught in Hawaii in 1985				Poor	48M/96F	No	Yes	Cheng
UCD Randombred Quail	Wild-type feather color pattern, unselected, randomly grouped in colony cages (2M/4F); reproduced every six to eight months.	Derived from stock imported from Japan and Taiwan (1950s and 1972)				Good	200+	No	Yes	Wilson
UMaryland Randombred Quail		Acquired from U Wisconsin/USDA in the 1970s; kept as closed flock for approximately 20 years				Good	100	No	With collaboration	Ottinger
UNL Wild-type Coturnix		Acquired from U Georgia-Athens				Fair: Hatch, regional, misc.	30-60 pairs	No	With collaboration	Beck
Selected-Behavioral trait										
Purdue Coturnix KGB	Selected for non-aggressive behavior starting in 1988.	Derived from Athens Control quail (U Georgia-Athens)				Good: Industry	10,000 birds	No	With collaboration	Muir
Selected-Growth trait										
Arkansas H10	Selected 18 generations for high 10-day body weight.	Derived from Arkansas RBC				Good	36M/36F	No		Anthony
Arkansas H17	Selected 18 generations for high 17-day body weight.	Derived from Arkansas RBC				Good	36M/36F	No		Anthony
Arkansas H28	Selected 18 generations for high 28-day body weight.	Derived from Arkansas RBC				Good	36M/36F	No		Anthony
Arkansas H40	Selected 18 generations for high 40-day body weight.	Derived from Arkansas RBC				Good	36M/36F	No		Anthony
Arkansas HL	Selected for high early body weight gain (10-17 days), low later body weight gain (17-28 days).					Good	36M/36F	No		Anthony
Arkansas LH	Selected for low early body weight gain (10-17 days), high later body weight gain (17-28 days).					Good	36M/36F	No		Anthony
Athens 52	High body weight selected.	Derived from a cross of Athens 51 and 53 (both selected 38 generations for high 4-week body weight)				Good	36M/36F	No	Yes	Burke
Athens 54	Low body weight selected.	Derived from a cross of two stocks, both selected 38 generations for low 4-week body weight				Good	36M/36F	No	Yes	Burke
Japanese quail genetic stocks										

Appendix 2 Table 2.127

Category

Japanese quail genetic stocks

Table 2.1 Stock information

Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserved	Available	Curator
Japanese quail (cont.)										
<u>Selected-Physiological trait (cont.)</u>										
	Athens L-PCHOL Selected for low blood plasma cholesterol for 37 generations; currently relaxed selection.	Derived from Athens Control quail				Good	30M/30F	No	Yes	Burke
	Louisiana High Stress Response Selected over ten years for high blood corticosteroid levels in response to stress.	Derived from Louisiana Randombred Quail				Good	60M/120F	No	Yes	Satterlee
	Louisiana Low Stress Response Selected over ten years for low blood corticosteroid levels in response to stress.	Derived from Louisiana Randombred Quail				Good	60M/120F	No	Yes	Satterlee
	Ohio HP Inbreeding coefficient (F)=0.36; selected 30 generations for increased plasma phosphorus in the female two weeks after start of lay.	Derived from Ohio R1				Poor	36M/36F	No	Yes	Nestor
	Ohio LP Inbreeding coefficient (F)=0.394; selected 30 generations for decreasing levels of plasma phosphorus in the female two weeks after start of lay.	Derived from Ohio R1				Poor	36M/36F	No	Yes	Nestor
	UBC RES Selected for resistance to atherosclerosis when fed high cholesterol diets.	Acquired from NCSU in 1988				Poor	24M/48F	No	Yes	Cheng
	UBC SUS Selected for susceptibility to atherosclerosis when fed high cholesterol diets.	Acquired from NCSU in 1988				Poor	48M/96F	No	Yes	Cheng

Table 2.1 Stock information

Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds	Cryo-preserv.	Available	Curator
Turkey (cont.)										
<u>Randombred (cont.)</u>										
Ohio RBC3	Inbreeding coefficient (F) approximately 0.033.	Derived from a cross of Ohio F Line with commercial turkey strains in 1986	Large White			Poor	36M/36F	No	Yes	Nestor
Wisconsin Broad-breasted Bronze	Old commercial-type turkey.	Derived from Tylor Bronze of Jerome Organization in N. Wisc.; kept as closed flock since 1969	Broad-breasted Bronze			Good	30M/200F	Semen, PGC	Yes	Wentworth
Wisconsin Broad-breasted White	Old commercial-type turkey.	Derived from Nicholas X Ralston synthetic meat cross developed at Washington State U; kept as closed flock since 1969	Broad-breasted White			Good	30M/200F	Semen, PGC	Yes	Wentworth
<u>Selected-Egg trait</u>										
NCSU Ohio-E	Selected for increased egg production.	Derived from Ohio-E; kept as closed flock	Large White			Good	10M/60F	No	Yes	Christensen
Ohio E Line	Inbreeding coefficient (F)=0.478; selected 38 generations for increased egg production.	Derived from Ohio RBC1	Large White			Poor	72M/72F	No	Yes	Nestor
<u>Selected-Growth trait</u>										
NCSU Ohio-F	Selected for increased body weight at 16 weeks.	Derived from Ohio-F; kept as closed flock	Large White			Good	10M/60F	No	Yes	Christensen
Ohio F Line	Inbreeding coefficient (F)=0.26; selected 30 generations for increased 16-week body weight.	Derived from Ohio RBC2	Large White			Poor	36M/72F	No	Yes	Nestor
Ohio FL Line	Inbreeding coefficient (F)=0.274; selected 17 generations for increased shank width.	Derived from Ohio F line	Large White			Poor	36M/54F	No	Yes	Nestor

Table 2.1 Stock information							
Category	Stock name and description	Origin and history	Breed	Genetic characteristics	Allele symbol	Status	Number of birds

Table 2.2. Stocks listed by country, state or province, institution, and curator.

Country—State/Province	Institution	Country—State/Province	Institution
Species/Category/Stock name		Species/Category/Stock name	
CANADA—BC University of British Columbia		CANADA—ON University of Guelph	
Curator: Kimberly Cheng		Curator: Robert J. Etches	
<u>Chicken</u>		<u>Chicken</u>	
Mutant-Developmental defect-Eye		Chromosomal variant	
UBC RC		Ottawa B-19/B-19 M13	Cryo. only
Mutant-Gene pool		Ottawa B-21/B-21 M13	Cryo. only
UBC-Minnesota Marker		Inbred	
Pure breed		Ottawa GF	Cryo. only
UBC-Minnesota Rhode Island Red		Ottawa GH	Cryo. only
<u>Japanese quail</u>		Ottawa M2	Cryo. only
Inbred		Ottawa WG	Cryo. only
UBC I		Ottawa XP	Cryo. only
Mutant-Color, eggshell		Pure breed	
UBC CE		Guelph Silkie	
UBC WE		Ottawa New Hampshire	Cryo. only
Mutant-Color, feather		Randombred	
UBC BH		Ottawa 10	Cryo. only
UBC C-CE		Ottawa 20	Cryo. only
UBC D		Ottawa 30	Cryo. only
UBC F-SB		Ottawa 5	Cryo. only
UBC H		Ottawa 7	Cryo. only
UBC RH		Selected-Egg trait	
UBC SI		Guelph Barred Plymouth Rock	
UBC W-WE		Guelph Commercial Leghorn Line	
UBC WB		Ottawa 1	Cryo. only
UBC Y		Ottawa 2	Cryo. only
Mutant-Developmental defect-Skin/feather		Ottawa 3	Cryo. only
UBC DF-MDF		Ottawa 4	Cryo. only
UBC PC-WB		Ottawa 8	Cryo. only
UBC RF-RH		Ottawa 9	Cryo. only
UBC RT		Selected-Growth trait	
UBC SB		Ottawa 21	Cryo. only
UBC SP		Ottawa 23	Cryo. only
Mutant-Uncategorized		Ottawa 25	Cryo. only
UBC H5		Ottawa 31	Cryo. only
Randombred		Selected-Immune trait	
UBC A		Ottawa 2R	Cryo. only
UBC B		Ottawa 3R	Cryo. only
UBC M		Ottawa 8R	Cryo. only
UBC N		Transgenic	
UBC NC		Ottawa TR	Cryo. only
UBC S		Uncategorized	
UBC WILD		Ottawa 6	Cryo. only
Selected-Growth trait		Ottawa 80	Cryo. only
UBC G-QM		Ottawa 90	Cryo. only
UBC QF		Ottawa N3	Cryo. only
UBC QM		Ottawa N4	Cryo. only
Selected-Physiological trait		Ottawa N8	Cryo. only
UBC RES		<u>Turkey</u>	
UBC SUS		Mutant-Reproductive defect	
		Guelph Parthenogenetic Turkey	

Table 2.2. Stocks listed by country, state or province, institution, and curator.

Country—State/Province	Institution	Country—State/Province	Institution
Species/Category/Stock name		Species/Category/Stock name	
CANADA—SK University of Saskatchewan		USA—AR University of Arkansas—Fayetteville	
Curator: Henry L. Classen		Curator: N.B. Anthony	
<u>Chicken</u>		<u>Selected-Growth trait</u>	
<u>Mutant-Neurological defect</u>		Arkansas H10	
Saskatchewan Epileptiform seizures		Arkansas H17	
<u>Pure breed</u>		Arkansas H28	
Saskatchewan Barred Plymouth Rock		Arkansas H40	
Saskatchewan Brown Leghorn		Arkansas HL	
<u>Turkey</u>		Arkansas LH	
<u>Pure breed</u>		<u>USA—AR University of Arkansas—Fayetteville</u>	
Saskatchewan Bronze Turkey		Curator: Gisela Erf	
<u>Waterfowl/Gamebird</u>		<u>Chicken</u>	
<u>Pure breed</u>		<u>Mutant-Immunological defect</u>	
Saskatchewan Pilgrim Goose		Arkansas Smyth Line B101	
Saskatchewan Rouen Duck		Arkansas Smyth Line B102	
<u>USA—AL Auburn University</u>		<u>Randombred</u>	
Curator: Wallace Berry	Other contact: Gayner R. McDaniel	Arkansas Brown Line B101	
<u>Chicken</u>		Arkansas Brown Line B102	
<u>Selected-Growth trait</u>		Arkansas Light Brown Leghorn B101	
Auburn Tibial Dyschondroplasia-High		<u>Selected-Immune trait</u>	
Auburn Tibial Dyschondroplasia-Low		Arkansas Progressor	
<u>USA—AL Auburn University</u>		Arkansas Regressor	
Curator: Sandra Ewald		<u>USA—AR University of Arkansas—Fayetteville</u>	
<u>Chicken</u>		Curator: John Kirby	
<u>Bloodtype-Gene pool</u>		<u>Chicken</u>	
Auburn DAX		<u>Mutant-Reproductive defect</u>	
<u>Bloodtype-MHC</u>		Arkansas Sd Line	
Auburn M		<u>USA—CA University of California—Davis</u>	
Auburn N		Curator: Ursula K. Abbott	Other contact: J.M. Pisenti
Auburn RM		<u>Chicken</u>	
Auburn RMH		<u>Mutant-Developmental defect-Face/limb</u>	
Auburn RN		UCD Cleft Primary Palate/Scaleless	
<u>Selected-Immune trait</u>		UCD Coloboma X 003	
Auburn R		UCD Diplopodia-1 X 003	
Auburn S		UCD Diplopodia-3 X 003	
<u>USA—AR University of Arkansas—Fayetteville</u>		UCD Donald-duck Beak/Scaleless	
Curator: N.B. Anthony		UCD Eudiplopodia X 003	
<u>Chicken</u>		UCD Limbless X 003	
<u>Pure breed</u>		UCD Polydactyly X 003	
Arkansas Giant Jungle Fowl		UCD Stumpy X 003	
<u>Randombred</u>		UCD Talpid-2 X 003	
Arkansas Randombred		UCD Wing-reduced X 003	
<u>Selected-Physiological trait</u>		UCD Wingless-2 X 331	
Arkansas Ascites Resistant		<u>Mutant-Developmental defect-Skin/feather</u>	
Arkansas Ascites Susceptible		UCD Ichthyosis X 003	
<u>Japanese quail</u>		UCD Naked-neck	Cryo. only
<u>Mutant-Color, feather</u>		UCD Scaleless-High	
Arkansas English White		UCD Scaleless-Low	
Arkansas White		<u>Mutant-Developmental defect-Spine/tail</u>	
<u>Randombred</u>		UCD Scoliosis	Cryo. only
Arkansas RBC			

Table 2.2. Stocks listed by country, state or province, institution, and curator.

Country—State/Province	Institution	Country—State/Province	Institution
Species/Category/Stock name		Species/Category/Stock name	
USA—CA University of California—Davis		USA—CA University of California—Davis	
Curator: Ursula K. Abbott		Curator: Barry Wilson	
Other contact: J.M. Pisenti			
Mutant-Gene pool		Chicken	
UCD Diplopodia-3/Scaleless High		Mutant-Physiological defect	
UCD Eudiplopodia/Limbless		UCD 413	
UCD Limbless/Stumpy		Randombred	
UCD Silkie cross		UCD 412	
UCD Talpid-2/Wingless-2		Japanese quail	
Mutant-Physiological defect		Randombred	
UCD Crooked-neck Dwarf		UCD Randombred Quail	
UCD Riboflavin Transfer Deficient			
Mutant-Uncategorized		USA—CT University of Connecticut	
UCD Crest/Hemoglobin-D	Cryo. only	Curator: Louis J. Pierro	
UCD Multiplex Comb	Cryo. only	Chicken	
Pure breed		Mutant-Developmental defect-Face/limb	
UCD Silkie		Storrs Chondrodystrophy	
		Storrs Creeper	
		Storrs Diplopodia-3	
		Storrs Diplopodia-5	
		Storrs Limbless	
		Storrs Micromelia-Abbott	
		Storrs Micromelia-Hays	
		Storrs Nanomelia	
		Storrs Perocephaly	
		Storrs Polydactyly	
		Storrs Talpid-2	
		Storrs Wingless-2	
		Mutant-Developmental defect-Skin/feather	
		Storrs Ottawa Naked	
		Storrs Ptilopody	
		Storrs Scaleless	
		Mutant-Developmental defect-Spine/tail	
		Storrs Dominant Rumplessness	
		Storrs Recessive Rumpless	
		Mutant-Physiological defect	
		Storrs Crooked-neck Dwarf	
		Storrs Muscular Dystrophy	
		Mutant-Uncategorized	
		Storrs Rose Comb	
USA—CA University of California—Davis		USA—DE University of Delaware	
Curator: Hans Abplanalp		Curator: Harold B. White III	
Chicken		Chicken	
Bloodtype-MHC-Inbred		Mutant-Physiological defect	
UCD 003		UDel Riboflavin Transfer Deficient	
UCD 253			
UCD 254			
UCD 312			
UCD 313	Cryo. only		
UCD 330			
UCD 331			
UCD 335			
UCD 336			
UCD 342			
UCD 361			
UCD 380			
UCD 386			
UCD 387			
Mutant-Immunological defect			
UCD 200			
Mutant-Physiological defect			
UCD 077			
Selected-Egg trait			
UCD 058			
UCD 082			
USA—CA University of California—Davis		USA—GA University of Georgia	
Curator: Mary E. Delany		Curator: William H. Burke	
Chicken		Chicken	
Chromosomal variant		Mutant-Physiological defect	
UCD-Cornell Mono-PNU		Athens-Canadian Dwarfs	
UCD-Cornell Trisomic		Randombred	
Inbred		Athens Randombred	
UCD 001		Athens-Canadian Randombred	
Mutant-Physiological defect			
UCD-Cornell Autosomal Dwarf			

Table 2.2. Stocks listed by country, state or province, institution, and curator.

Country—State/Province	Institution	Country—State/Province	Institution
Species/Category/Stock name		Species/Category/Stock name	
USA—GA University of Georgia		USA—IL Northern Illinois University	
Curator: William H. Burke		Curator: W. Elwood Briles	
Selected-Immune trait		Chicken	
Athens AR		Bloodtype-Gene pool	
Japanese quail		NIU B haplotypes	
Ranombred		NIU B-haplotype Recombinants	
Athens Control Quail		NIU Male Breeder Alloantigen Reservoir	
Selected-Growth trait		NIU Segregating Male Breeder Line	
Athens 52		Bloodtype-MHC	
Athens 54		NIU Female Breeder Parent Stock B19	
Athens 56		NIU Female Breeder Parent Stock B2	
Athens P-line		NIU Female Breeder Parent Stock B5	
Athens T-line		Waterfowl/Gamebird	
Selected-Immune trait		Bloodtype-Gene pool	
Athens AR3.0		NIU Bobwhites	
Selected-Physiological trait		NIU Pheasants	
Athens H-PCHOL		USA—IL University of Illinois—Urbana	
Athens L-PCHOL		Curator: Carl Parsons Other contact: Bob Leeper	
USA—IA Iowa State University		Chicken	
Curator: Susan J. Lamont		Pure breed	
Chicken		UI-Urbana Columbian	
Bloodtype-MHC-Inbred		UI-Urbana New Hampshire	
ISU 19-13		USA—IN Purdue University	
ISU 19-15.1		Curator: W.M. Muir	
ISU 8-15.1		Chicken	
ISU G-B1		Selected-Behavioral trait	
ISU G-B2		Purdue KGB	
ISU GH-1		Purdue MBB	
ISU GH-13		Japanese quail	
ISU GH-15.1		Selected-Behavioral trait	
ISU HN-12		Purdue Coturnix KGB	
ISU HN-15		USA—LA Louisiana State University—Baton Rouge	
ISU M15.2		Curator: Daniel G. Satterlee	
ISU M5.1		Japanese quail	
ISU Sp21.2		Ranombred	
Mutant-Uncategorized		Louisiana Ranombred Quail	
ISU S1-19H		Selected-Physiological trait	
ISU S1-19L		Louisiana High Stress Response	
ISU S1-1H		Louisiana Low Stress Response	
ISU S1-1L		USA—MA University of Massachusetts—Amherst	
USA—IA National Animal Disease Center		Curator: J. Robert Smyth Jr	
Curator: Richard Rimler		Chicken	
Turkey		Mutant-Developmental defect-Eye	
Ranombred		UMass Pink Eye	
NADC Turkey		Mutant-Immunological defect	
		UMass Smyth Line	
		Pure breed	
		UMass Light Brown Leghorn	
		Ranombred	
		UMass Brown Line	

Table 2.2. Stocks listed by country, state or province, institution, and curator.

Country—State/Province	Institution	Country—State/Province	Institution
Species/Category/Stock name		Species/Category/Stock name	
USA—MA University of Massachusetts—Amherst		USA—MI USDA-ARS-Avian Disease and Oncology Laboratory	
Curator: J. Robert Smyth Jr		Curator: Larry D. Bacon	
<u>Turkey</u>		Other contact: Laura Parks	
Mutant-Developmental defect-Eye		<u>Inbred</u>	
UMass Glaucoma		ADOL 6C.7A ReCon	
USA—MD University of Maryland—College Park		ADOL 6C.7B ReCon	
Curator: Mary Ann Ottinger		ADOL 6C.7C ReCon	
<u>Japanese quail</u>		ADOL 6C.7D ReCon	
<u>Randombred</u>		ADOL 6C.7F ReCon	
UMaryland Randombred Quail		ADOL 6C.7G ReCon	
USA—MI USDA-ARS-Avian Disease and Oncology Laboratory		ADOL 6C.7I ReCon	
Curator: Larry D. Bacon		ADOL 6C.7J ReCon	
Other contact: Laura Parks		ADOL 6C.7K ReCon	
<u>Chicken</u>		ADOL 6C.7L ReCon	
<u>Bloodtype-MHC</u>		ADOL 6C.7M ReCon	
RPRL-Cornell JM-N		ADOL 6C.7N ReCon	
RPRL-Cornell JM-P		ADOL 6C.7P ReCon	
<u>Bloodtype-MHC-Inbred</u>		ADOL 6C.7R ReCon	
RPRL 15.15-5		ADOL 6C.7S ReCon	
RPRL 15.6-2		ADOL 6C.7T ReCon	
RPRL 15.7-2		ADOL 6C.7V ReCon	
RPRL 15.C-12		ADOL 6C.7W ReCon	
RPRL 15.N-21		ADOL 6C.7X ReCon	
RPRL 15.P-13		<u>Transgenic</u>	
RPRL 15.P-19		ADOL Trans-ALVE6	
RPRL 15I5		<u>Waterfowl/Gamebird</u>	
RPRL 7I1		<u>Pure breed</u>	
RPRL Line 0		ADOL Pekin Duck	
<u>Endogenous virus</u>		USA—NC North Carolina State University—Raleigh	
RPRL 15B1		Curator: Vern L. Christensen	
<u>Endogenous virus-Inbred</u>		<u>Chicken</u>	
RPRL 100B		<u>Pure breed</u>	
RPRL 6I3		NCSU Barred Plymouth Rock	
RPRL 7I2		NCSU Rhode Island Red	
RPRL Reaseheath Line C		<u>Turkey</u>	
		<u>Pure breed</u>	
		NCSU Black Spanish	
		NCSU Bronze	
		NCSU Slate	
		<u>Randombred</u>	
		NCSU Ohio-RBC1	
		NCSU Ohio-RBC2	
		<u>Selected-Egg trait</u>	
		NCSU Ohio-E	
		<u>Selected-Growth trait</u>	
		NCSU Ohio-F	
		<u>Waterfowl/Gamebird</u>	
		<u>Pure breed</u>	
		NCSU Brown China	
		NCSU Pilgrim	

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Country—State/Province	Institution	Country—State/Province	Institution
Species/Category/Stock name		Species/Category/Stock name	
USA—NC North Carolina State University—Raleigh		USA—NY Cornell University	
Curator: Muquarrab Qureshi		Curator: K.A. Schat	
<u>Chicken</u>		<u>Chicken</u>	
Bloodtype-MHC-Inbred		Bloodtype-MHC	
NCSU GB-1		Cornell N2a	
NCSU GB-2		Cornell P2a	
Randombred		Selected-Immune trait	
NCSU-Cornell K Strain		Cornell S13	
USA—NE University of Nebraska—Lincoln		<u>Waterfowl/Gamebird</u>	
Curator: Mary Beck		Pure breed	
<u>Chicken</u>		Cornell White Pekin	
Mutant-Neurological defect		USA—OH Ohio State University—OARDC	
UNL Paroxysm		Curator: Karl E. Nestor	
<u>Japanese quail</u>		<u>Japanese quail</u>	
Randombred		Randombred	
UNL Wild-type Coturnix		Ohio R1	
USA—NH University of New Hampshire		Selected-Growth trait	
Curator: Robert Taylor Jr		Ohio HW	
<u>Chicken</u>		Ohio HW-HP	
Bloodtype-Gene pool		Ohio HW-LP	
UNH 105		Ohio LW	
Bloodtype-MHC		Selected-Physiological trait	
UNH 192		Ohio HP	
Bloodtype-MHC-Inbred		Ohio LP	
UNH 6.15-5		<u>Turkey</u>	
UNH 6.6-2		Randombred	
UNH-UCD 003		Ohio RBC1	
UNH-UCD 003.R1		Ohio RBC2	
UNH-UCD 003.R2		Ohio RBC3	
UNH-UCD 003.R3		Selected-Egg trait	
UNH-UCD 003.R4		Ohio E Line	
UNH-UCD 003.R5		Selected-Growth trait	
UNH-UCD 003.R6		Ohio F Line	
UNH-UCD 386		Ohio FL Line	
UNH-UCD 387		USA—OH Ohio State University—OARDC	
UNH-UCD 3C.1		Curator: Sandra G. Velleman	
USA—NY Cornell University		<u>Chicken</u>	
Curator: Rodney R. Dietert		Mutant-Physiological defect	
<u>Chicken</u>		Ohio Low-score Normal	
Selected-Egg trait		Ohio Muscular Dystrophy	
Cornell K Strain		USA—OR Oregon State University—Corvallis	
USA—NY Cornell University		Curator: Thomas F. Savage	
Curator: James Marsh		<u>Chicken</u>	
<u>Chicken</u>		Mutant-Gene pool	
Mutant-Immunological defect		OSU Dwarf Leghorn	
Cornell Obese (B-13)			
Mutant-Physiological defect			
Cornell Sex-linked Dwarf			
Randombred			
Cornell C Specials (B13)			

Table 2.2. Stocks listed by country, state or province, institution, and curator.

Country—State/Province	Institution	Country—State/Province	Institution
Species/Category/Stock name		Species/Category/Stock name	
USA—PA Pennsylvania State University		USA—WI University of Wisconsin—Madison	
Curator: Guy F. Barbato		Curator: Bernard C. Wentworth	
<u>Chicken</u>		<u>Japanese quail</u>	
Selected-Growth trait		Mutant-Gene pool	
PSU-Athens RB 14-42H (HiK)		Wisconsin Japanese Quail	
PSU-Athens RB 14-42L (LoK)			
PSU-Athens RB 14H		<u>Turkey</u>	
PSU-Athens RB 14L		Pure breed	
PSU-Athens RB 42H		Wisconsin Midget White	
PSU-Athens RB 42L		Randombred	
		Wisconsin Broad-breasted Bronze	
		Wisconsin Broad-breasted White	
USA—VA Virginia Polytechnic Institute and State University			
Curator: Paul B. Siegel		Other contact: E. Ann Dunnington	
<u>Chicken</u>			
Selected-Growth trait			
Virginia Body Weight-High			
Virginia Body Weight-Low			
Selected-Immune trait			
Virginia Antibody Line-High			
Virginia Antibody Line-Low			
USA—WI University of Wisconsin—Madison			
Curator: James J. Bitgood		Other contact: John F. Fallon	
<u>Chicken</u>			
Chromosomal variant			
Wisconsin Chromosomal Rearrangements			
Inbred			
Wisconsin Ancona			
Wisconsin Leghorn line UW-Sp2			
Mutant-Color, feather			
Wisconsin Autosomal Albino			
Mutant-Developmental defect-Eye			
Wisconsin Blind/Cataract			
Wisconsin Pink-eye			
Wisconsin Pop-eye			
Mutant-Developmental defect-Face/limb			
Wisconsin Ametapodia			
Wisconsin Limbless			
Wisconsin Talpid-2			
Wisconsin Wingless-2			
Mutant-Developmental defect-Skin/feather			
Wisconsin Tardy Feathering			
Mutant-Neurological defect			
Wisconsin Pirouette			
Mutant-Reproductive defect			
Wisconsin Double Oviduct Line			
Wisconsin Restricted Ovulator			
Mutant-Uncategorized			
Wisconsin Sex-linked Skin Color			
Randombred			
Wisconsin Leghorn			
Wisconsin Leghorn line UW-6X			
Wisconsin New Hampshire			

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Top right: Wild-type Japanese quail (UCD Randombred Quail) used in a wide range of experimental studies. Female (right) and male (left). Photograph courtesy of J. Clark, University of California–Davis)

Center left: *Fawn* mutation in the Japanese quail (UBC F-SB). (Photograph courtesy of K. Cheng, University of British Columbia)

Center right: Male giant Japanese quail (UBC G-QM). More than double the size of unselected quail, this line was developed by intensive selection for increased six-week body size in the females. (Photograph courtesy of K. Cheng, University of British Columbia)

Right: Modern commercial Large White turkeys. (Photograph courtesy of R.A. Ernst, University of California–Davis.)



